Public Health Reports

VOLUME 56 MARCH 21, 1941 NUMBER 12

IN THIS ISSUE

U.S. PUBLIC HEALTH SERVICE
1798

FEDERAL SECURITY AGENCY

UNITED STATES PUBLIC HEALTH SERVICE

THOMAS PARRAN, *Surgeon General*

DIVISION OF SANITARY REPORTS AND STATISTICS

CHARLES V. AKIN, *Assistant Surgeon General, Chief of Division*

The PUBLIC HEALTH REPORTS, first published in 1878 under authority of an act of Congress of April 29 of that year, is issued weekly by the United States Public Health Service through the Division of Sanitary Reports and Statistics, pursuant to the following authority of law: United States Code, title 42, sections 7, 30, 93; title 44, section 220.

It contains (1) current information regarding the prevalence and geographic distribution of communicable diseases in the United States, insofar as data are obtainable, and of cholera, plague, smallpox, typhus fever, yellow fever, and other important communicable diseases throughout the world; (2) articles relating to the cause, prevention, and control of disease; (3) other pertinent information regarding sanitation and the conservation of the public health.

THE PUBLIC HEALTH REPORTS is published primarily for distribution, in accordance with the law, to health officers, members of boards or departments of health, and other persons directly or indirectly engaged in public health work. Articles of special interest are issued as reprints or as supplements, in which forms they are made available for more economical and general distribution.

Requests for and communications regarding the PUBLIC HEALTH REPORTS, reprints, or supplements, should be addressed to the Surgeon General, United States Public Health Service, Washington, D. C. Subscribers should remit direct to the Superintendent of Documents, Washington, D. C.

Librarians and others should preserve their copies for binding, as the Public Health Service is unable to supply the general demand for bound copies. Indexes will be supplied upon request.

UNITED STATES GOVERNMENT PRINTING OFFICE, WASHINGTON : 1941

For sale by the Superintendent of Documents, Washington, D. C.

Price 5 cents. Subscription price $2.50 a year

CONTENTS

Public Health Reports

Vol. 56 • MARCH 21, 1941 • *No. 12*

WAR AND INFECTIOUS DISEASE [1]

By CLARA E. COUNCELL, *Junior Statistician, United States Public Health Service*

CONTENTS

The waging of war has always been attended by increases in the prevalence of disease. The rapid and extensive spread of infection is to be expected under the conditions brought about by the struggles between nations. The concentration and movement of large bodies of men from various parts of the world; the limitless hardships, with fatigue, general malnutrition, famine and exposure; and the lack of medical care, sanitation, and personal hygiene often experienced by civilians and soldiers alike provide the fuses for the explosion of widespread epidemics. Refugees and captured and returning prisoners are important instruments in the transmission of disease from enemy to enemy and to all civilian groups. While certain types of sickness have accompanied armies throughout the centuries, there have nevertheless been some notable changes in the prevalence and severity of wartime affections. It is only in comparatively recent wars that more men have been lost from military action than from disease. The ratio of the disease death rate to the battle death rate among United States troops was 7 to 1 in the Mexican War and 5 to 1 in the Spanish War. The Germans in the Franco-Prussian War of 1870 and the Japanese and Russians in the Russo-Japanese War of 1904 show the first records in which the mortality for the wounded was higher than for those stricken with infectious disease.

The true degree of relationship between war conditions and the unusual prevalence of disease must be, to some extent, a matter of conjecture. It is doubtful whether the pandemic of syphilis at the end of the fifteenth century can be entirely explained by its dissemination by soldiers throughout Italy, France, and Germany. It has been suggested that the spread of virulent smallpox throughout

[1] From the Division of Public Health Methods, National Institute of Health. The writer is greatly indebted to Dr. S. D. Collins for valuable suggestions and criticism.

Europe after 1870 cannot be explained wholly by the events of the Franco-Prussian War. The prevalence of cerebrospinal meningitis in 1918–19 and 1939–40 may be interpreted as evidence of a wave in epidemicity that would have manifested itself in the absence of the contributing factors present in wartime. It is, however, obvious that usually mild diseases may flare up with unparalleled activity and, fanned by the abnormal circumstances of living, may rage with uncontrollable fury. It is difficult to judge, currently or in retrospect, the extent of specific causes of illness, as the general onslaught of infections makes precise diagnosis difficult if not entirely impossible. The similarity and overlapping of the symptoms of various diseases suggest simultaneous attacks of more than one of the usually recognized entities and may easily result in the incorrect allocation of many cases. While this may be especially true of the epidemics described by the early historians, it is not limited to the experience of past centuries. There is evidence, for example, that at the time of the war of 1914–18 cases of typhoid fever were undoubtedly included in the horde of sufferers from the influenza pandemic (*15b*). It is impossible to show the true prevalence of the epidemic diseases of early or even of modern wars, and the number of cases reported must often represent only a fraction of the total. No attempt has been made here to relate epidemic spread to specific troop movements and similar wartime events, but some of the records available have been utilized to show which affections appear to have been of primary importance and to what degree. The data here collected are but a few examples from the awesome toll of wartime sickness.

WAR AND INFECTIOUS DISEASE BEFORE 1914

Knowledge of early pestilences is dependent entirely upon the historian's powers of estimation and description. Only a blurred picture can be obtained of the true character and incidence of the great waves of fatal illness that decimated the nations involved in early wars. Ignorance concerning the transmission of the common diseases and of the principles of health protection made the people particularly vulnerable to mass attack. Nevertheless, from all of the evidence presented, it appears that much of the extensive damage wrought in the past can be ascribed to diseases identifiable today.

Typhus fever was prevalent through ancient times and the Middle Ages (*18*). After Fracastori's clear description early in the sixteenth century, this disease is readily traceable in wars in southern Europe and in Germany and Austria-Hungary. During the Thirty Years' War (1618–48) in which most of Europe became involved, it accompanied the armies in the East. It was present during the War of the Polish Succession (1733–35), and occurred among troops in the Seven Years' War (1756–63) when Austria, France, Russia, and Sweden were

aligned against Prussia and England. The French Revolution of 1789 and the Napoleonic Wars (1805–14) were followed by the most severe epidemic in the history of Europe. Typhus spread generally even to sections far removed from siege and battle, although the disease was always more severe in the war area. It again played great havoc in the Russo-Turkish War of 1877–78. The simultaneous occurrence of epidemics of typhus and of relapsing fever has long been recognized.

Galen has described a pestilence of unmistakable smallpox. Beginning in the Roman Army in A. D. 166, it spread over the whole of Italy and into Gaul, resulting in the death of more than half of the population and almost all of the troops (*10g*). The Arabian physician Rhazes described the disease accurately in the ninth century and it seems to have been reintroduced again and again from the East by the returning Crusaders. Smallpox was apparently brought into the New World by the discoverers of America and took a large toll of the native tribes. In the following centuries severe epidemics were frequent in the Eastern and Western Hemispheres until they moderated after the introduction of vaccination at the end of the eighteenth century. Armies and civilians suffered greatly from smallpox during the American Civil War (1861–65). At the time of the Franco-Prussian War it was very prevalent among the French, but few of whom had been vaccinated, although there was little among the better protected German Army. War prisoners carried the epidemic to Germany where a large part of the population was not vaccinated, and no nineteenth century epidemic could compare in extent and virulence with that which raged after 1870 throughout Germany, Belgium, Switzerland, the Netherlands, Austria, England, and America, to which it was brought by immigration (*16*).

Typhoid fever has not been described as such in the records of early wars but it has undoubtedly been one of the leading scourges. It was apparently the most fatal disease of the American Civil War, and was very common in the Franco-Prussian War when it spread throughout France, in the Russo-Turkish War of 1877–78, and in the Boer War of 1899–1902 between the British and Dutch. During the Spanish-American War (1898) the incidence rate was 142 per 1,000 per annum (*15b*). Large numbers of soldiers contracted typhoid, diarrhea, and dysentery in the Russo-Japanese War of 1904.

Dysentery is described in Hindu records of 3,000 years ago, and was well known to the early Greeks and Romans. It was differentiated from diarrhea by Hippocrates. The armies of the Crusades, in the thirteenth century, suffered greatly from dysentery. Records indicate that this disease was especially widespread during the Thirty Years' War and the Franco-Prussian War and killed thousands of French and British soldiers in the Crimean War (1853–56) involving Turkey, Great Britain, and France against Russia. While difficulties of dif-

ferentiation make figures for dysentery particularly inaccurate, some indication of its importance may be gleaned from the following reports of its incidence and fatality in the past *(10a)*:

War	Reported number of—	
	Cases	Deaths
Franco-German (1870-71) (German Army)	38, 652	2, 380
South African campaign (1899-1902)	38, 108	1, 342
Sino-Japanese War (1894)	155, 140	38, 094

Diarrhea and dysentery of an unusually fatal character were responsible for large numbers of deaths among American Civil War soldiers.

Records of war fought in regions where malaria was endemic indicate its high and fatal attack rate. It was of unusual severity when Napoleon besieged Mantua, a town of northern Italy, in 1796–97 and during the Italian Revolutionary War of 1859. Over half of the American Civil War troops acquired malaria *(16)*.

The parallelism of the war of 1914–18 and the influenza pandemic have made this disease of significance as one that may be exacerbated by wartime conditions. Accounts of early epidemics refer to the occurrence of a disease that was apparently identical with or closely allied to influenza. Some historians so identify the pestilence of 412 B. C., mentioned by Hippocrates and Livy and this diagnosis may be applied to numerous epidemics occurring after the sixth century, such as the English sweating sickness of 1486. Pandemics of influenza readily identifiable as such occurred in the sixteenth and eighteenth centuries, with recurrences in 1830–33, 1836–37, 1847–48, and 1889–90 *(19)*.

Two of the diseases which formerly took a large toll in times of war have apparently become of less importance. Bubonic plague in the twelfth century devastated the soldiers of Frederick Barbarossa in his Italian campaigns, as well as the population of Rome and surrounding territory occupied by the armies. It was perhaps the most prevalent disease of the Thirty Years' War and was severe in all Europe throughout the Middle Ages. It was epidemic during the Russo-Turkish Wars of 1768–74 and 1827–29, but in later years it has not played a major role among war pestilences. With generally improved sanitation cholera also has declined in importance. It was exceptionally common at the time of the Crimean War, when it spread over all of France, and scattered from the Crimea over a large part of eastern Europe. At the time of the German-Austrian War of 1866 about 100,000 persons died of cholera in Germany. Extremely low incidence followed the practice of inoculation by the Japanese in 1904 and by

the Greek Army in the Balkan War (1912–13) between Turkey and the allied armies of Bulgaria, Greece, Montenegro, and Serbia.

INFECTIOUS DISEASE AMONG CIVILIANS DURING AND AFTER THE WAR OF 1914–18

The experience of the war of 1914–18 has the recurring theme of epidemic diseases of the past. The distress of earlier times was paralleled by the experience of Russia in the period during and after the war and revolution. The resulting famine throughout Russia, unequaled since the time of the Thirty Years' War, was accompanied by cholera, dysentery, malaria, typhoid, typhus, and relapsing fever. Throughout the country there was a lack of food, drugs, and fuel; many hospitals were closed, and in others all types of patients were crowded together, often more than one person in a bed. A constant flow of refugees filled the roads, the stations, and all means of transportation, and epidemics followed the lines of railways and waterways. The suffering of the people was intense, especially in the eastern and southern parts of the country, and their situation became increasingly desperate in the post-war years.

Cholera appeared in epidemic form in the Ukraine, a southern district of European Russia, in December 1920, flared high in 1921, when 176,885 cases were officially notified, and again in 1922. Before the war there were annually about 80,000 cases of typhus and 30,000 of relapsing fever reported in Russia, with typhus ranging from a low of 36,887 in 1897 to about 180,000 cases in the famine years of 1892 and 1909. Typhus became increasingly serious after 1917 and in the epidemic of 1919–20 there were nearly 5,000,000 cases of typhus and 1,260,000 of relapsing fever recorded for the civilian population (*12b*). A recession in 1921 was followed by great increases in typhus and relapsing fever, as well as cholera, in 1922. Typhoid and dysentery were also extremely prevalent. Tarassevitch quotes the following figures for the years 1918 through 1921 (*12c*):

	1918	1919	1920	1921
Typhoid fever	109, 624	252, 066	424, 481	406, 389
Dysentery	59, 750(?)	137, 169(?)	324, 389	220, 093

The queries are his and he comments that the figures are certainly very incomplete. He concludes that typhoid fever and the dysentery group were of greater prevalence than before the war but in comparison with that of typhus and relapsing fever their increase is only very slight.

Malaria was one of the most widespread diseases in the country, but was so incompletely reported that it is practically impossible to

estimate its extent. It was apparently the unanimous opinion of doctors that malaria greatly increased throughout the country during and after the war years.

Figures available for the first 9 months of 1921 indicate the comparative prevalence and completeness of reporting in European Russia. The Red Army constituted only about 1 percent of the total population. While preventive inoculation and the special conditions of army life complicate any comparison, there is nevertheless strong evidence that all of these diseases, and particularly relapsing fever, were grossly underreported for civilians.

Number of cases reported in European Russia, Jan.–Sept. 1921 (12a)

	Red Army	Railways [1]	Other cases (European Russia civilian population) [2]	Total	Red Army as percent of total
Typhus	61, 984	19, 751	330, 178	411, 913	15.0
Typhoid fever	16, 248	14, 619	150, 445	181, 312	9.0
Dysentery	16, 049	12, 447	162, 549	191, 045	8.4
Relapsing fever	258, 670	36, 003	270, 313	564, 986	45.8
Asiatic cholera	2, 836	19, 328	100, 893	123, 057	2.3

[1] It is not clear whether this refers to travelers or to railway personnel and their families.
[2] Excluding Ukrainia.

In 1918 influenza spread over Russia. There were marked increases in tuberculosis, in other infections of the respiratory tract, and the venereal diseases, as well as milder outbreaks of infectious jaundice, encephalitis lethargica, scabies, tinea, and trachoma. Apparently all affections except the common communicable diseases of children showed greater distribution and prevalence.

The organization of strong public health defenses upon the Polish and southern borders aided in preventing the spread of disease throughout Europe by refugees escaping from the famine zones of Russia. By 1918 there was a great migration of Polish prisoners of war and civilian refugees well under way. About 468,000 persons returned to Poland in 1921 and in each month of the summer and autumn between 50,000 and 60,000 persons passed through the quarantine station at Baranowicze, near the Russian border of Poland. During 1 month almost 1,500 deaths occurred among persons arriving at the station *(12a)*. Records of hospital admissions among the repatriates showed an even greater incidence of dysentery, malaria, and relapsing fever than of typhus, in addition to a high proportion of cases of measles and pulmonary tuberculosis.

From the beginning of the war of 1914–18 all Poland had been a battlefield and epidemics followed on the heels of the many armies with which it was overrun. In Poland, as well as Russia, typhus was continually present. It appeared in epidemic form in 1916 and reached a peak in 1919 and 1920. The number of cases declined

during the summer months but rose with new intensity each fall and increased greatly over those of pre-war years (*11a*):

Year:	*Number of deaths from typhus fever*
1911	426
1916	3,480
1917	3,776
1918	7,655
1919	19,891
1920	22,565

In Congress Poland and Galicia, post-war districts of eastern and southern Poland, there was an average of about 2,000 cases per year reported for the period 1905 through 1911. In 1919–20 the annual incidence had risen to about 200,000 cases, or 100 times the number reported in the earlier years. It has been estimated that in these sections, in the month of January 1920, there were at least 40,000 cases of typhus, with about 100,000 cases in all Poland. After some decline typhus flared up again in the winter of 1921–22, after which the recession was continuous for 10 years or so. The typhus increase was accompanied by a rise in the number of cases of relapsing fever, with about 14,000 cases reported in 1921.

Many other diseases, perhaps originating in Bolshevik prisoners' camps, spread to the army and the civilian population of Poland. Between 1895 and 1915 the records show only 3 instances of the appearance of cholera in Galicia, with 459 deaths in one outbreak in 1895, 19 in 1896, and 1 imported case in 1910. In 1915, coincident with the advance of the Russian Armies, there was a violent flare-up with more than 30,000 cases and 17,252 deaths (*11a*).

There were recurring epidemics of cholera; dysentery, typhoid fever, and smallpox were everywhere. Measles and scarlet fever also accompanied many of the refugee children (*11b*).

Number of cases notified in Poland in 1919 and 1920 (*11a*)

	1919	1920
Typhus fever	234,938	157,612
Dysentery	15,304	32,938
Typhoid fever	12,246	20,868
Smallpox	1,862	3,746

The summer and autumn of 1920 witnessed a considerable increase in the incidence of dysentery. It had been particularly virulent in 1914–15 in western Galicia and in 1917 in Congress Poland. The infection was apparently transported from this area, with the German troops, to south Hungary and Serbia.

Early in 1919 investigations of the serious epidemic conditions of eastern Europe were initiated by the League of Red Cross Societies, and, with the organization of the Epidemic Commission of the League

of Nations in 1920, determined efforts were made to control the epidemic crises.

Of the rest of Europe, only Serbia suffered from epidemics such as those of Russia and Poland. Weakened by the Balkan Wars immediately preceding the war of 1914–18, Serbia was an easy victim of epidemic disease. Typhus spread to all parts of the country from the wounded, sick, and imprisoned Austrians, particularly at Valjevo, in northern Serbia. At the same time relapsing fever was widespread and typhoid fever increased. Records of incidence are lacking, but the Serbian typhus outbreak of 1915 probably attacked at least 1 in every 5 persons, with a case fatality of from 30 to 70 percent, and in less than 6 months over 150,000 people died of typhus (*18*). In the spring of 1915 Serbia organized a vigorous campaign against typhus, with aid from the United States, France, Great Britain, Belgium, and Holland; and by August the epidemic had waned, not to reappear.

Statistics for the post-war period indicated that the epidemic prevalence of typhus and relapsing fever accompanied repatriates through the eastern area (*12b*), in Estonia, Latvia, Lithuania, Rumania, Turkey, and Yugoslavia (the Serb-Croat-Slovene Kingdom). The peak of the epidemic in eastern Europe occurred in 1919 and 1920. It has been estimated that among the 115 million people of European Russia and the Ukraine there were 3,000,000 cases in 1920 and among the combined population of about 55 million in Rumania, Poland, Lithuania, Latvia, and Estonia there were 225,000 cases of typhus.

Estimated number of cases of typhus fever (13a)

Year	European Russia and the Ukraine	Other parts of eastern Europe
1920	3,000,000	225,000
1921	550,000	62,000
1926	50,000	6,500
1931	15,000	4,000

Among the Austrian civilian population more than 13,000 cases of typhus occurred in 1915 and over 12,000 in 1916. Cases of typhus and relapsing fever also appeared in some districts of Germany, chiefly among prisoners of war. Although there was general infestation with lice among soldiers and civilians on the western front, for some reason that has never been evident these areas were strikingly free of typhus and relapsing fever.

In the years during and after the war of 1914–18 smallpox flared up in all parts of the world. After the passage of a compulsory vaccination law in Germany in 1874 the disease was relatively quiescent until it became epidemic in north Germany in 1917. The 2,400 cases then reported were the largest number on record. In 1915–16 smallpox

was violently epidemic in Austria-Hungary, where about 50,000 cases occurred. More than 42,000 of these cases were in the two Provinces of Galicia and Bukowina, from which the infection was carried to the larger cities, including Vienna, Prague, and Budapest (*10c*).

Italy had an annual average of 700 cases for 1914–16, but in 1919 and 1920 there were respectively 34,365 and 26,543 cases (*8*). In England and Wales there were only 7 cases of smallpox reported in 1917. In 1918 there were 63 cases and in the next 5 years the numbers were 311, 280, 336, 973, and 2,504. The recrudescence was not limited to the war zone, for the United States had an annual average of 28,000 cases for 1909–14, but there were 56,332 cases reported in 1919, 96,684 in 1920, and 102,787 cases with 641 deaths in 1921. The registration area of British India reported great increases in 1919 and 1920 and Australia was invaded by smallpox between 1914 and 1918 and again in 1921.

Tuberculosis mortality showed marked increases during and immediately following the war of 1914–18. In a world where control measures were at a standstill, during a period when living conditions would in every way encourage the spread of infection, and when the influenza toll was great, the death rate from tuberculosis showed the dramatic rise that was to be expected.[2] Not only in Germany, England, Belgium, the Netherlands, Italy, and Austria but also in the United States and Japan the wartime peak was evident (*21*). If data were collected for France and other warring countries, it is to be expected that they would show a similar picture.

In 1917 the Polish cities of Warsaw, Cracow, and Lodz had tuberculosis death rates of, respectively, 840, 908, and 1,164 per 100,000 population (*4*). In Vienna this rate increased from 278 in 1914 to 490 in 1919. In Berlin the pre-war rate of 156 in 1913 was almost doubled by the 1917 rate of 292. In the 15 years preceding the war, the death rate from all forms of tuberculosis in the Prussian district of Germany fell from 219 per 100,000 population in 1899 to 142 in 1914. During the war years it rose to 230 in 1918 and was again high in the inflation period 1922–23. In Belgium the death rate increased from 118 per 100,000 population in 1913 to 245 in 1918. The rapid decline of tuberculosis after the war has been explained by the fact

[2] The increase in tuberculosis reflects generally impaired nutrition in the years during and after the war. The limitation of this paper to infectious disease neglects any relationship between war and other types of sickness. Periods of famine also result in the disturbance of growth, particularly in very young children, and in the increased prevalence of the deficiency diseases, such as beriberi, pellagra, rickets, and scurvy. In the Moscow industrial and the central agricultural districts of Russia there were 5,317 cases of scurvy reported in 1914–15 and 55,972 in 1920–21. Increases in child and maternal mortality have been noted. During and after the war of 1914–18 there was a large amount of disability from nervous and mental disorders in the fighting forces. Opinion is at present divided regarding the effect of air raids upon the incidence of mental disorders in the civilian population. It has been suggested that the stresses and strains of war may raise the morbidity and mortality from diseases of the heart and blood vessels. Industrial fatigue is an important element of wartime. In general, anxiety, fatigue, and the lowering of the standard of living may reduce resistance to disease and may result in the impairment of mental and physical functions and organs of the body.

that many acutely infectious cases were wiped out in the influenza pandemic, a situation somewhat comparable to the decline of leprosy in Europe after the ravages of the Black Death.

After the beginning of hostilities malaria reappeared in the Emden district, in the northeastern corner of Germany, where it had been rare since 1890. The increase was ascribed chiefly to the neglect of canals and drainage systems. Dysentery is known to have been epidemic in Austria and in Germany, especially in Prussia. During the first 7 months of 1917 there were more than 4,000 cases in Austria, while in Prussia there were about 13,000 cases among civilians. The epidemic was apparently introduced from the armies, in which it was even more severe (*10c*).

Cerebrospinal meningitis [3] was widespread and fatal, not only in the war zone but also in other parts of the world. Figures for Austria show 3,226 cases with 1,601 deaths in the 4 years 1915–18, inclusive. In the last 6 months of 1915 alone there were 222 cases with 107 deaths. In England this disease maintained a prevalence hitherto unequaled. In the wartime epidemic period of 1914–18 there were 6,450 cases reported among civilians in England and Wales and 4,238 cases among military personnel (*3b*).

Outbreaks of encephalitis lethargica were noted among British and French civil and military populations in 1917–18. The disease swept various European countries at different times and attained its highest incidence in and after 1920. In the United States and most of Europe a decline was evident after 1924 (*13b*).

Association with influenza has been noted in the occurrence of cerebrospinal meningitis, encephalitis lethargica, and other epidemic diseases of the central nervous system. At the end of the war of 1914–18 influenza completely overshadowed all other diseases in importance, and in extent and virulence was comparable to the plagues of early history. The occurrence of the pandemic of 1918–19 may have been coincidental with, but was certainly exacerbated by, the conditions of wartime and its aftereffects. Both incidence and mortality are matters of estimate. The marked increases in the death rate for all causes were everywhere greater than the rise in influenza and pneumonia death rates. The excess noted in the mortality from bronchitis, pulmonary tuberculosis, heart disease, certain puerperal causes, and nephritis in the United States suggests that the effect of epidemic influenza upon the population cannot be shown by consideration of influenza-pneumonia mortality alone. Jordan (*9*) has estimated the total mortality from the influenza pandemic to have been nearly 22,000,000 persons, distributed as follows:

[3] Wherever this category is discussed it presumably refers to disease due to meningococcus infection, as in item No. 6 of the International List of Causes of Death (1938 revision), but because of confusion and incomplete statements of etiology the content may vary and it may include a number of cases not of meningococcic origin.

Total mortality, influenza pandemic, 1918

	Number of deaths
North America	1,076,000
South America	327,000
Europe	2,163,000
Asia	15,757,000
Australia and Oceania	965,000
Africa and Madagascar	1,354,000
Total	21,642,000

While these figures represent only general approximations it is probable that they do not overestimate the true picture.

Definitive figures for venereal disease are hard to obtain. There was apparently an increase in the civilian population as well as in the armies of Europe (*3a, 7, 10b*).

INFECTIOUS DISEASE IN THE FIGHTING FORCES IN THE WAR OF 1914–18

European armies—war of 1914–18.—The armies in western Europe were not harassed by typhus, relapsing fever, and cholera, nor after the early stages of the war by smallpox, dysentery, or typhoid fever. During the war of 1914–18, in contrast to earlier conflicts, more soldiers were killed in battle than died from disease.

Typhus and relapsing fever assumed epidemic proportions only in the armies of Russia, Serbia, and Poland. On both eastern and western battlefronts the general infestation with lice was held responsible for the numerous cases of trench fever which, though of a low fatality, caused much sickness. About one-half of 1 percent of the German troops on the Polish front had cholera, and about 12,000 cases occurred in the army in September 1914. Inoculation is credited with having effected the low fatality rate of 10 percent (*5*). Cholera was prevalent in Turkey and among the fighting forces of Russia, Serbia, and Austria-Hungary, but occurrence among other troops was rare.

The troops for whom vaccination was compulsory were generally free from smallpox. The Austro-Hungarian Army, only partially protected, had many cases. Amebic dysentery was imported to France by the colonial troops, and British forces in the Dardanelles suffered severely with diarrheal diseases. Dysentery was the common disease in the German Army, with more than 155,000 cases and a fatality rate of about 12 percent. It was especially severe among the troops in the eastern campaign (*6*).

Typhoid fever, war of 1914–18 (15b)

Country	Cases		Deaths		Case fatality
	Number	Rate per 1,000	Number	Rate per 1,000	
					Percent
United States [2]	1,572	0.4	233	0.05	14.8
Great Britain	6,807	1.0	260	.04	3.8
France [1]	124,991	14.9	15,211	1.81	12.2
Italy [1 2]	58,451	6.2			
Belgium [2]	3,217	3.6	523	.57	16.3
Germany [2]	112,364		11,405		10.2
Austria	171,601		17,399		10.1

[1] Includes paratyphoid fever.
[2] Reported cases for the United States Army are for the entire period 1914–19, including time before entry into the war. Italy did not enter the war until 1915 and the United States not until the spring of 1917. Figures for the United States and Belgium include cases occurring during 1919. The figures for Germany and Italy do not include cases occurring in 1914.

In 1914 and 1915 the French Army suffered greatly from typhoid fever and in January 1915 there were 13,993 cases with 2,210 deaths. With increasing inoculation the prevalence dropped, until in December 1916 only 323 cases were reported (*17a*). During the first 2 years of the war the French Army had about 110,000 cases of typhoid and paratyphoid fever in comparison with 2,000 for the last 2 years, after the widespread use of triple vaccine.

In the malarious regions of Taranto, in southern Italy, Macedonia, in northern Greece, in Asia Minor, and in East Africa, troops were seriously affected. The British Macedonian Expeditionary Force in 1917 had 71,412 admissions for malaria, with 228 deaths, out of a force of 182,000 men. In the 1917 East African campaign the 50,000 men had over 72,000 admissions, with 499 deaths (*10d*).

Epidemic jaundice was common among armies in all parts of Europe and was very prevalent in Gallipoli at the end of 1915. Cerebrospinal meningitis, as well as influenza and pneumonia, were widespread among all troops after the fall of 1917 and the toll of diseases of the respiratory system was especially high. The French Army in particular suffered severe losses from tuberculosis. Out of a total force of about 8,400,000 men mobilized during the war, 111,000 French soldiers were discharged for tuberculosis between August 1914 and December 1918. Rist (*17b*) has, however, estimated that out of the 86,000 soldiers discharged for this cause during the first year of the war less than 20 percent were really tuberculous. MacNalty (*3b*) reports that about 35,000 men were invalided out of the British services on account of tuberculosis attributable to or aggravated by conditions of service. There was also a rise in the tuberculosis rate for the German Army during the war years.

The belief that venereal disease was much more prevalent among the armies of the war of 1914–18 than in previous times was substantiated by the records of the British, French, German, Austro-Hungarian, and American Armies. One of the most difficult of problems lay in the practice of the men who deliberately acquired venereal disease in order to absent themselves from the trenches for treatment (*10e*).

The experience of the war of 1914–18 shows that the incidence of infectious diseases among the troops was controlled to an extent far greater than in the past by sanitation, hygiene, and preventive inoculation. The German Army was strikingly successful in combating epidemic infections, with a record of 1,531,048 deaths from wounds and 155,013 from disease during 1914–19. Waldman (*20*) contrasts this proportion of 1 death from disease to each 10 for wounds to the less favorable ratios of earlier wars.

United States Army—war of 1914–18 (*15*).[4]—In the United States Army, cholera, typhus fever, smallpox, and malaria played exceptionally unimportant roles among the infections prevalent during the war of 1914–18. Among the communicable diseases the largest numbers of reported primary admissions were for influenza, venereal diseases, mumps, and measles; and influenza, tuberculosis, measles, and cerebrospinal meningitis were leading causes of death. Epidemics were on the whole more extensive in the training camps in this country than in the American Expeditionary Forces.

[4] All data quoted in this section were obtained from the volumes of the Medical Department of the United States Army in the World War unless other references are specified.

Admissions and days lost from duty for certain infectious diseases, U. S. Army,[1] *Apr. 1, 1917–Dec. 31, 1919*

	Primary admissions		Days lost from duty	
	Number	Annual rate per 1,000	Number	Noneffective rate per 1,000[2]
Respiratory diseases[3]	1,125,401	272.6	17,042,838	11.3
United States	749,004	335.1	9,368,434	11.5
Europe	335,484	201.4	7,306,906	12.0
Influenza	791,907	191.8	10,676,172	7.1
United States	533,649	238.7	6,146,574	7.5
Europe	228,461	137.2	4,296,815	7.1
Bronchitis	255,148	61.8	3,287,643	2.2
United States	169,426	75.8	1,543,152	1.9
Europe	76,975	46.2	1,669,261	2.8
Pneumonia (all)	78,346	19.0	3,079,023	2.0
United States	45,929	20.5	1,678,708	2.1
Europe	30,048	18.0	1,340,830	2.2
Venereal diseases (all)	357,969	86.7	6,804,818	4.5
United States	284,742	127.4	4,745,450	5.8
Europe	57,195	34.3	1,748,067	2.9
Gonococcus infection	251,899	61.0	3,903,303	2.6
United States	211,638	94.7	2,867,491	3.5
Europe	31,199	18.7	891,492	1.5
Syphilis (all)	67,026	16.2	1,927,901	1.3
United States	51,528	23.0	1,345,961	1.6
Europe	12,680	7.6	511,657	.8
Chancroidal infection	39,044	9.5	973,614	.6
United States	21,576	9.6	531,998	.6
Europe	13,316	8.0	344,918	.6
Mumps	230,356	55.8	3,884,147	2.6
United States	141,628	63.4	2,276,544	2.8
Europe	81,853	49.1	1,501,222	2.5
Measles	98,225	23.8	1,877,944	1.2
United States	85,398	38.2	1,600,798	2.0
Europe	9,168	5.5	229,745	.4
Dysentery (all), diarrhea, enteritis and colitis	92,512	22.4	1,060,229	.7
United States	39,854	17.8	232,241	.3
Europe	48,202	28.9	793,972	1.3
Tuberculosis of lungs	33,249	8.0	3,385,053	2.2
United States	27,274	12.2	2,636,722	3.2
Europe	4,877	2.9	677,169	1.1
German measles	17,378	4.2	211,645	.1
United States	16,167	7.2	197,330	.2
Europe	579	.4	8,505	(4)
Malarial fevers (all)	15,555	3.8	194,529	.1
United States	10,510	4.7	130,673	.2
Europe	950	.6	20,477	(4)
Scarlet fever	11,675	2.8	498,190	.3
United States	9,038	4.0	382,628	.5
Europe	2,370	1.4	106,877	.2
Diphtheria	10,909	2.6	317,050	.2
United States	5,884	2.6	144,452	.2
Europe	4,860	2.9	168,100	.3
Meningitis, cerebrospinal (epidemic)	4,831	1.2	268,164	.2
United States	2,878	1.3	150,386	.2
Europe	1,848	1.1	114,110	.2
Chickenpox	1,757	.4	31,534	(4)
United States	1,208	.5	21,443	(4)
Europe	388	.2	7,582	(4)
Typhoid fever	1,529	.4	109,374	.1
United States	546	.2	28,587	(4)
Europe	885	.5	76,649	.1
Smallpox	853	.2	24,275	(4)
United States	780	.4	21,890	(4)
Europe	24	(4)	1,110	(4)
Trench fever	798	.2	34,098	(4)
United States	11	(4)	674	(4)
Europe	786	.5	33,402	(4)

[1] Total mean annual strength=4,128,479; United States (including Alaska)=2,235,389; Europe (excluding Russia)=1,665,796; other (Philippine Islands, Panama, etc.)=227,294.

[2] Noneffective rate$=\frac{\text{total days lost}\times 1{,}000}{\text{total mean annual strength}}\div 365$.

[3] Respiratory diseases include influenza, bronchitis, lobar and broncho-pneumonia and pneumonia, unclassified.

[4] Rate per 1,000 less than 0.1.

The accompanying table shows, for important infectious diseases, the number of primary admissions and days lost from duty,

with rates, for the United States Army from April 1, 1917, through December 31, 1919, in all areas; in the United States, including Alaska; and in Europe, excluding Russia. The total mean annual strength of these forces was 4,128,479, with 2,235,389 in the United States and 1,665,796 in Europe.

The number of admissions refers to admissions to sick report and includes not only men admitted to hospitals but also those treated either when excused from duty or for ailments probably affecting their future military fitness, as well as all deaths, discharges, and retirements. Only one cause of admission was recorded. Deaths and days lost were ascribed to the original cause of admission, irrespective of subsequent complications. There was a total of 3,515,464 admissions for sickness in the United States Army in the period under discussion, with 62,681,428 days of disability. Rates shown are annual ratios per 1,000 mean strength. The noneffective rate shows the average number of men absent from duty for each day of the year. To obtain it the total days lost were divided by the total mean annual strength, and the quotient was divided by 365 days.

Because of their relative unimportance Asiatic cholera, with a total of 17 admissions and 7 deaths, and typhus fever, with 42 admissions and 3 deaths, are not included in the table.[5] Smallpox accounted for only 853 admissions and 141 deaths. Before the war of 1914–18 the Army smallpox rate had always risen abruptly with the start of war, but for this period both admission and death rates showed little change from the figures for preceding years. Malaria was responsible for a total of 15,555 admissions, of which 10,510 occurred in the Army in the United States. The total number of 36 deaths from malaria is a very low figure for an army in wartime.

Typhoid fever, a primary disabling factor in the Civil and Spanish-American Wars, appears low on the list of important infections of the war of 1914–18. A sharp decline in incidence in the Army, starting in 1910, corresponds with the introduction of compulsory inoculation. For typhoid fever as for smallpox no extreme increase was noted in the wartime records in comparison with those for preceding years. Many of the 546 admissions in the United States occurred prior to inoculation. The American Expeditionary Forces, reporting 885 admissions, had supposedly all been inoculated at some time, but the lapse of time since inoculation, less sanitary surroundings, and increased exposure to infection increased the risk of attack.

Infections of the respiratory tract supplanted the pestilences of earlier wars as leading causes of sickness and death. Influenza, bronchitis, and pneumonia combined were responsible for 1,125,401 admissions and 44,270 deaths, with an annual admission rate of 273

---

[5] See *15b*, page 483; in *15c*, pages 86 and 134, 2 admissions and 1 death are reported.

per 1,000, a death rate of 11 per 1,000, and a case fatality of 4 percent. Together they accounted for over 17,000,000 days lost from duty. Influenza was apparently more frequent and more fatal in the camps in this country than in the troops abroad. Increased prevalence in the fall of 1918, culminating in the October peak, appeared to run a parallel course in both forces. Data for the Army as a whole show a subsequent decline followed by recurring waves in January and February, June and July, and December, 1919.

Of great importance among causes of sickness and disability was the group of venereal diseases, responsible for 357,969 admissions to sick report, or slightly more than 10 percent of the total recorded, and with a loss of nearly 7,000,000 days from duty. The comparatively high rate for the Army in the United States is explained by the fact that the majority of these cases were brought into the service at the time of the draft, while an effort was begun toward the end of 1917 to keep men with venereal disease from service abroad. There was a steady decline in the reported venereal disease cases in the American Expeditionary Forces up to September 1918, but in subsequent months the rate seems to have increased.

Dysentery, diarrhea, and enterocolitis are best considered as a group because of the difficulties of differentiation, especially in the field. In the Civil War this group was reported to be the leading cause of illness (*14*). They were of less importance in the Army during 1917–19 than in preceding conflicts but were still of considerable moment, especially when it is remembered that many minor cases do not appear in the reported figures.

Measles was of serious consequence, as in the Civil War, not only because of its frequency but also because of the complicating pneumonia largely responsible for the 2,370 deaths ascribed to measles as the primary cause of admission. This disease is an extremely sensitive index of rapid expansion of the Army, with a characteristic flare-up in incidence upon the assembling in camps of large numbers of young men, many from rural areas and previously unexposed to the risk of infection. Mumps had a widespread occurrence and was of great moment in the American Expeditionary Forces. Its importance is particularly evident in the high total number of days lost from duty as a result of the numerous cases. The 17 days lost per case were comparable to those of 19 for measles and 18 for chickenpox, but the number of cases was much greater.

Diphtheria, scarlet fever, chickenpox, German measles, and poliomyelitis were not of relatively great effect upon the United States Army in 1917–19. They were of some note chiefly from the standpoint of noneffectiveness. Only scarlet fever ranked high among deaths from all causes. Infectious jaundice was reported responsible for 452 admissions and 15 deaths.

There were several diseases of which little or no previous mention had been made that commanded major interest in the period under discussion. Cerebrospinal meningitis was of relatively low incidence and was sporadic rather than epidemic in its occurrence in the Army, but its extremely high mortality made it of great importance as a cause of death in the United States forces. The case fatality varied in different camps from 9 to 63 percent and there were 1,836 deaths ascribed to this primary cause.

A relationship between the occurrence of influenza and of encephalitis lethargica has been noted. This type of encephalitis was recognized in 1917 in central Europe and appeared subsequently in other European countries and in the United States. The cases were not numerous but their spread was rapid and wide. The total occurrence in the Army is not known, but at least 20 cases, with 4 deaths, have been identified from clinical records.

Trench fever had apparently never been described as such prior to the war of 1914–18. It was responsible for 798 admissions and 2 deaths in the United States Army, with 786 of the admissions reported in the European forces. Vincent's disease, or trench mouth, was very rare in this Army before the arrival of the American troops in the European trenches. The total of 6,000 reported admissions among enlisted men in the United States and Europe is not representative of the extent of the occurrence of this comparatively mild affection.

There were 33,249 admissions for pulmonary tuberculosis in the entire Army, with 2,240 deaths. As an additional safeguard against tuberculosis, reexamination of the entire United States Army for this infection was begun in July 1917. Only some 40,000 men already sent abroad and some units leaving hurriedly at later times were not included in the reexamination. The signs of tuberculosis leading to rejection, including interpretation of X-ray findings, were carefully defined. The findings are summarized in the following table:

Reexaminations for pulmonary tuberculosis

	Number examined	Cases	
		Number	Per 1,000
Officers' training camps	53, 905	195	3. 6
Aviation service	38, 835	62	1. 6
Regular Army in the field	190. 396	1, 444	7. 6
Coast Artillery Corps	40, 396	297	7. 4
National Guard	446, 517	[1] 4, 907	11. 0
Troops of National Army (first draft)	361, 314	2, 435	6. 7

[1] Estimated from number examined and rate quoted.

Up to March 1918 a total of 1,200,990 men had been reexamined and 9,648 recommended for discharge for pulmonary tuberculosis, a total

of 8 per 1,000. In the period of demobilization, from November 1918 up to June 30, 1919, there were 2,500,662 men examined of whom only 1,356, or 0.54 per 1,000, were found to be tuberculous.

Ayres (*2*) compares the annual number of deaths per 1,000 troops for the United States Army in the war of 1914–18 and in preceding conflicts to show a striking decline in the deaths from disease:

	Annual number of deaths per 1,000 troops	
	Disease	Battle
Mexican War, 1846–48	110	15
Civil War (North), 1861–65	65	33
Spanish War, 1898	26	5
War of 1914–18:		
Overseas (to Nov. 11, 1918)	19	53
United States and France (to May 1, 1919)	15	13

He ascribes the improvement in the death rate from disease to the services of a highly trained medical personnel, compulsory typhoid inoculation, improved sanitation, and provision of adequate hospital facilities.

United States Navy—war of 1914–18 (*1*).—The wartime increase in the size of the naval forces was accompanied by the widespread occurrence of mumps and measles. There were many cases of cerebrospinal meningitis, with high fatality, and tuberculosis and pneumonia were leading causes of death. Lobar and broncho-pneumonia were responsible for more deaths in the Navy than all the other communicable diseases combined. The Navy as a whole suffered little from typhoid fever, scarlet fever, and diphtheria. The comparatively low venereal disease rates early in the war were ascribed to the excitement and active duty, the appeal to patriotism, and enforced prohibition of alcohol for men in uniform. The absence of these factors after the signing of the armistice may at least partially explain the increase in the rate noted for 1919.

Admission rates in the entire Navy for certain infectious diseases for the active war years and for 1916 and 1919 were as follows:

	Admission rates per 1,000 strength			
	1916	1917	1918	1919
Cerebrospinal fever (meningococcus)	([1])	2.1	1.7	0.4
Diphtheria	0.7	.8	3.6	2.8
Measles	7.6	30.8	13.7	3.6
Mumps	10.6	39.8	35.4	19.7
Scarlet fever	1.3	2.7	2.4	2.4
Tuberculosis (all forms)	4.1	3.2	4.8	4.7
Typhoid fever	.2	.3	.2	.2
Venereal diseases	149.0	88.7	70.2	111.6

[1] Not given.

Because of the special conditions of ship life the Navy afloat constitutes a unit hardly comparable to the armies and civilians of countries at war. The fleet is largely protected by a quarantine period of observation from the introduction of infection by men drafted for duty. The barracks, depots, and training stations of the Navy are, however, more exposed to infection, for as in Army training camps the stream of new entries, the aggregation of susceptible material, and the contact with civilians invite epidemic invasion. During the war of 1914–18 the incidence of infectious diseases was generally greater among the men of the United States Navy ashore than among those afloat. This is illustrated by the following figures for influenza, bronchitis, and pneumonia in the year 1918:

	Number	Rate per 1,000		Number	Rate per 1,000
Total:			Bronchitis:		
Forces afloat [1]	50,219	162.0	Forces afloat [1]	4,520	14.6
Forces ashore	96,227	496.5	Forces ashore	9,216	47.5
Influenza:			Pneumonia (all forms):		
Forces afloat [1]	42,947	138.6	Forces afloat [1]	2,752	8.9
Forces ashore	77,457	399.6	Forces ashore	9,554	49.3

[1] Naval forces afloat and expeditionary forces (including marines) combined. The strength of the entire Navy for 1918 was 503,792; that of the forces afloat and expeditionary forces (including marines) combined was 309,974; and that of the forces ashore in the United States was 193,818.

INFECTIOUS DISEASE IN THE PRESENT WORLD WAR

Continental Europe—Present world war.—Up to the present, mild influenza and cerebrospinal meningitis have been the most notable infections occurring in warring nations, with typhus fever showing increased activity in eastern Europe. Cyclic increases in cerebrospinal meningitis have been observed at intervals of from 8 to 12 years. A general decline in incidence, beginning after the war of 1914–18, was succeeded by marked increases in 1928–29. The recession from this peak continued until about 1934. In 1936 the disease was at the maximum of a new upward curve in the United States and a rise was observed in Italy and Greece. This rise was evident in 1937 in Poland, Turkey, Rumania, and Yugoslavia, and in 1938 in Bulgaria, Germany, England, and Scotland. In 1939 and the early part of 1940 the occurrence of the disease was extremely widespread in the latter countries as well as in Austria-Hungary, Slovakia, Yugoslavia, and Switzerland. The number of cases reported seems generally to have been without precedent, at least in recent years.

In Germany the notifications of cerebrospinal meningitis numbered 1,826 (2.7 per 1,000 population) in 1938 and 5,046 (7.5 per 1,000) in 1939. Hungary had an annual average of 52 cases from 1921 to 1928, whereas in 1939 there were 395 cases reported. Yugoslavia had 731 cases in 1939 in comparison with the 85 to 155 cases per year

for the preceding 12 years. Bulgaria had less than 50 cases a year from 1934 through 1936, but in 1939 there were 669. The figures available through March 1940 show a progressive decline in the United States since 1936 and do not indicate increased prevalence in Belgium, the Netherlands, or the Scandinavian countries. The current outbreaks show a widespread dispersion of sporadic cases, comparatively small seasonal variation, and a decline in case fatality.

Number of reported cases of cerebrospinal meningitis (13c)

Country	First 8 weeks		Country	First 8 weeks	
	1939	1940		1939	1940
England and Wales	287	2,336	Switzerland	4	164
Scotland	96	542	Hungary	27	270
Germany [1]	786	1,052	Yugoslavia	147	525

[1] Excluding Austria and the Sudeten area.

There was an increase of generally mild influenza in England and Wales, Scotland, Ireland, Germany, Hungary, Denmark, and Switzerland as well as in the United States. The outbreak in the winter of 1939–40 was not of the magnitude of the influenza epidemics immediately preceding it, in 1937, 1933, 1929, and 1927. The recorded data showed no unusual occurrence in Finland, Norway, Sweden, or the Netherlands.

The incidence of typhus fever declined for the 10 years after 1921, but more recently there has been a recrudescence in Poland, Rumania, and Russia, the chief European foci of this disease. In 1938 there were 7,295 cases of typhus reported from Europe (excluding the U. S. S. R.), of which 2,748 cases were in eastern Poland and 1,603 in Bessarabia, a far-eastern district of Rumania that had been a part of Russia. During the winter of 1939–40 some increase in typhus in comparison with the previous year was observed in Bessarabia, southern Yugoslavia, and parts of Turkey, Bulgaria, and Hungary. Complete figures were not available for Russia, nor for Poland where typhus accompanied the refugee movements. The disease has also appeared in some sections of Germany where it has not been endemic, notably in East Prussia and in Polish Silesia. In March 1940, an outbreak occurred in Mecklenburg, a district of northern Germany bordering on the Baltic, with 23 reported cases and 6 deaths. No increases were observed in the first quarter of 1940 in Slovakia, Bohemia, Latvia, Estonia, or Finland.

Typhus fever cases reported in Europe, 1937–39 inclusive (13d)

Country	Number of cases			Country	Number of cases		
	1937	1938	1939		1937	1938	1939
Germany	0	0	0	Poland	3,501	3,566	[1] 3,140
Bulgaria	160	91	129	Portugal	67	37	32
Spain			[1] 67	Rumania	4,949	2,254	1,024
Finland	1			Czechoslovakia	82	33	[1] 3
France	1	1	([1])	Turkey	667	450	463
Greece	97	93	[1] 96	Russia:			
Hungary	22	5	57	R. S. F. S. R. in Europe	[1] 18,744		
Ireland	5		5	White Russia	[1] 3,338		
Latvia	3	5	4	Ukraine	[1] 345		
Lithuania	110	124	154	Yugoslavia	908	638	411
Netherlands	1						

[1] Incomplete.

An outbreak of dysentery comparable to that of the war of 1914–18 occurred in the German Army during the Polish campaign of 1939. Polish refugees in Rumania and Hungary are reported to have suffered from typhus fever, malaria, and respiratory diseases.

Comparison of the total reported cases of typhoid and paratyphoid fevers for 1938 and 1939 shows no sizable increase in any country for which data are available, with the single exception of Finland. Here the notifications rose from 699 in 1938 to 1,048 in 1939. Out of a total of 15 countries in the war zone, 7 showed some increase in 1939 while 8 reported fewer cases than in 1938, as did Australia, Japan, and the United States.

Cases of typhoid and paratyphoid fevers reported in certain countries in 1938 and 1939 (13e)

Country	Number of cases		Country	Number of cases	
	1938	1939		1938	1939
Sweden	440	508	Germany (excluding Austria and the Sudeten area)	6,152	5,449
Finland	699	1,048	Austria	1,624	1,877
Netherlands	410	458	Belgium	286	351
France	4,120	4,373	Italy	41,330	30,024
Bohemia and Moravia	[1] 1,607	[1] 1,159	Yugoslavia	5,612	4,568
Slovakia	[1] 2,124	[1] 1,400	Hungary	7,399	7,117
Bulgaria	2,911	2,418	United States	14,248	12,808
Rumania	6,505	4,468	Australia	347	224
Japan	48,204	43,125			
England	1,321	1,520			

[1] Incomplete.

England—Present world. war.—After the war of 1914–18 England initiated and strengthened many public health measures, including maternal, child health, tuberculosis, and venereal disease programs, insurance medical service, and supervision of school children. At the end of 1938 the health record was good and improvement was indicated by the decline in mortality since 1919. The general death rate had dropped from 13.8 per 1,000 population to 11.6, the infant mortality rate from 89 per 1,000 live births to 53, the maternal mor-

tality rate from 4.4 per 1,000 live births to 3.1, the tuberculosis death rate from 126 per 100,000 to 60, and the typhoid death rate from 1.6 to 0.4 (*10f*).

The state of war existing since September 1939 might well be expected to result in an increased prevalence of infectious diseases. The mass migrations of large groups of the population were in particular viewed with pessimism. No such movement of the people of the British Isles had been known since the time of the Great Plague of 1665, when London was emptied of about two-thirds of its population. In the early fall of 1939 about 1,270,000 persons were moved, including 735,000 school children, 166,000 mothers and other adults with 260,000 young children, 12,000 expectant mothers, in addition to teachers and helpers and the blind or crippled. It was estimated that by the middle of the following January over 87 percent of the evacuees had returned home (*3c*).

On September 1, 1939, the evacuation of mothers and children began, from urban to rural areas and later from the south and east coasts to inland sections. The change in environment, with the intermediate massing in reception areas, the closing of schools and termination of school health activities, and the possible introduction of infection to previously unexposed groups provided excellent fuel for epidemics. It was thought that the migrants might readily introduce the common communicable diseases among the more susceptible rural children, while the infections carried by milk and water, especially typhoid and dysentery, would find fresh victims among the newcomers. The unpasteurized milk of the country, rare for city children, was a possible agent for the transmission of scarlet fever, diphtheria, undulant fever, and tuberculosis.

The health problem of the bomb shelters was intensified by the fact that many people slept in the close quarters originally provided only as temporary refuge during daylight raids. Although these shelters accommodate only a small proportion of the total population, large numbers of people are nevertheless involved. The opportunities for the transmission of disease were thus greatly enhanced (*3e*). Not only overcrowding but also lack of sanitation and sanitary supervision of heating and ventilation were thought to invite the spread of typhoid, dysentery, and the respiratory diseases.

Additional foci of tubercle infection resulted from the limitation in the hospitalization of the tuberculous to provide facilities for expected air-raid casualties. An unusually severe winter, with a shortage of fuel, and the black-out, both resulting in difficulties of ventilation, also encouraged the spread of respiratory infections. These particular factors augmented the health hazards of war resulting from mobilization, changes in dietary habits, nervous strain, lack of sleep, and increased fatigue, particularly among the industrial workers.

Reports of the incidence of infectious disease since the outbreak of the war have shown no such general increase as was feared, especially in the common communicable diseases of childhood. In the first 2 months of the sunny autumn during which most of the migration of school children took place, there was apparently less epidemic disease than in 2 corresponding months of peacetime in 1938. Improvement in the health of the child population was generally noted. Epidemics seemed to be controlled by the increased watchfulness, the closing of schools, and wide dispersal of children, rather than encouraged by the diffusion of carriers.

There were some localized outbreaks of diphtheria, scarlet fever, whooping cough, diarrhea, and dysentery, but the total incidence of infectious diseases was low, with the notified cases of diphtheria, poliomyelitis, and scarlet fever all lower than in 1938. Returns for the first 7 months of the war showed an almost complete absence of deaths from diphtheria, whooping cough, and measles. Whooping cough remained low both in prevalence and fatality. An expected biennial epidemic of measles did not materialize. Only chickenpox, and, to a greater extent, German measles, attained epidemic proportions. The freedom from childhood infections which characterized the early days of wartime in England was not continued, for the fall of 1940 brought increases in diphtheria, measles, scarlet fever, whooping cough, and poliomyelitis in comparison with the previous year, although in most cases these diseases were not as prevalent as in 1938.

In June 1940 there was a rise in the notifications of typhoid fever and by the end of July the reported cases were four times as numerous as in the corresponding week of 1939. No differentiation was made between typhoid and paratyphoid, but the mildness of the cases led to the conclusion that the latter predominated. Reports from local sources also indicated that outbreaks of paratyphoid have been fairly numerous and extensive.

The incidence of respiratory infections was somewhat higher than usual but there was a great drop in the number of pneumonia deaths, attributed at least in part to the use of the sulfanilamide compounds. Deaths from tuberculosis showed an increase.

Influenza and cerebrospinal fever were both extremely widespread. Influenza is not notifiable but some indication of its prevalence may be obtained from the number of deaths ascribed to this cause in the 126 great towns of England and Wales, which include over half of the total population, and from the cases of primary and influenzal pneumonia recorded for the entire country. The deaths from influenza reported for the 6 weeks ending March 2, 1940, were 416, 350, 514, 521, 629, and 512; these are higher than the average figures for this season of the year. In each of these 6 consecutive weeks over 2,000 cases of primary and influenzal pneumonia were recorded. The

numbers of influenza deaths and of pneumonia cases rose abruptly at the end of 1939 and continued high until the end of March. During the 17 weeks from early December through late March there were 4,685 deaths ascribed to influenza in the large towns, which may be compared with 3,009 in the corresponding weeks of 1938–39; 7,726 in 1936–37, and 10,660 in 1932–33 (*10i*). The normal age distribution, with old people particularly affected, suggested unusual prevalence but not severity.

Cerebrospinal fever in England and Wales (3d)

Year	Number reported		Year	Number reported	
	Cases	Deaths		Cases	Deaths
1913	305	232	1927	469	430
1914	315	396	1928	412	438
1915	2, 566	2, 203	1929	650	588
1916	1, 306	1, 368	1930	661	632
1917	1, 465	1, 651	1931	2, 152	1, 440
1918	798	926	1932	2, 087	1, 213
1919	848	694	1933	1, 668	942
1920	583	533	1934	1, 079	729
1921	411	416	1936	864	617
1922	344	360	1936	967	635
1923	300	284	1937	1, 112	698
1924	397	310	1938	1, 258	652
1925	402	354	1939	1, 508	----------
1926	384	365	1940 [1]	5, 093	----------

[1] From weekly returns of Registrar-General (corrected), first 13 weeks of year; the provisional figure for the year 1940 is about 12,500.

Previous notifications of cerebrospinal fever in England and Wales have never approached the 1939–40 figures. While the fatality rate was low, the cases were very numerous and scattered over the country during a long period after the rise in January. The epidemic continued into the summer and its course suggested the possibility of an even higher incidence this winter. In the first 10 weeks of the year, there were 3,558 cases reported, with a maximum in March, in comparison with 1,508 for all of 1939, 1,258 in 1938, and 3,500 in the 1915–16 epidemic. The case fatality varied greatly in different parts of the country but was on the whole much less than that of 1914–18, when the fatality was about 60 percent among civilians and sometimes reached as high as 80 percent; in the 1939–40 epidemic the average was closer to 10 or 12 percent.

In a review of Britain's mortality, by age, for the first 7 months of the war (September 1939–March 1940, inclusive) Stocks (*10h*) found that in the age group under 5 years there had been a fall in the death rate from pneumonia, bronchitis, diarrhea, and whooping cough. There were no material changes in the figures for children 5 through 14 years of age. Adults 15 through 44 showed an increased mortality during the winter period from heart disease, respiratory tuberculosis, and the respiratory diseases, with the exception of pneumonia, which

showed no marked increase. Among persons over 45 there was a great increase in mortality, particularly notable for heart disease, the respiratory affections, and traffic accidents.

According to an Associated Press dispatch for January 7, 1941, Sir William Jameson, chief medical officer of the Health Ministry, announced increases for England and Wales in the number of reported cases of cerebrospinal meningitis, pneumonia, typhoid fever, and dysentery for the year 1940. There were about 12,500 cases of cerebrospinal meningitis in 1940 as against 1,500 in 1939, but the fatality was low. Reported pneumonia cases were 42,000 in 1939 and 46,000 in 1940; typhoid fever, 1,500 in 1939 and 2,800 in 1940; and dysentery, 4,170 in 1938, falling to 963 in 1939 but reaching 2,500 in 1940. Diphtheria and scarlet fever showed a decline.

Up to the end of March 1940, the health of the Army had been good except for the epidemics of influenza, cerebrospinal fever, and German measles. The occurrence of cerebrospinal fever was sporadic and showed a fatality markedly lower than that of the war of 1914–18. In the first 6 months of the war a total of 371 cases had been reported in the troops at home and abroad. The general physical condition of the troops was considered to be better than in 1914–18 and it was felt that their food was better and that the use of the mobile baths should greatly lessen skin and parasitic infections. Although by February 1940 some treatment centers were reporting a material increase in the number of new cases of venereal disease, knowledge of treatment has greatly increased in the last few years and the prevalence in the defense forces was not considered comparable to that in the early stages of the last war when it has been estimated that about 400,000 men in the British Army of approximately 5,000,000 were infected.

The British feel that the prophylaxis against smallpox, tetanus, and typhoid fever, subject to the soldier's consent, should control the incidence of these diseases, while the new chemotherapy is expected to reduce the hazards of gonorrhea, cerebrospinal meningitis, and pneumonia.

Up to the present time respiratory infections are the objects of special concern in England, and colds, influenza, pulmonary tuberculosis, and cerebrospinal fever are particularly to be guarded against.

SUMMARY

The history of wars before the twentieth century is characterized by accounts of the great ravages of typhus fever, smallpox, typhoid fever, dysentery, malaria, cholera, and bubonic plague. In modern warfare, influenza and other respiratory affections, cerebrospinal meningitis, and venereal diseases are apparently of outstanding importance. While the historic pestilences remain lurking dangers, controlled chiefly by the unremitting application of the principles of

sanitation and immunology, it is unlikely that they will again be responsible for most of the mortality of wartime.

Infections invariably killed more soldiers than did bullets until, in the Franco-Prussian War of 1870, the German Army recorded an excess of deaths from battle. In the Russo-Japanese War of 1904 both sides maintained the higher proportion of deaths from wounds. In spite of the toll of influenza, the war of 1914–18 was the first in which United States fighting forces participated with fewer deaths from sickness than from battle, although for the entire Army, at home and abroad, the deaths from disease were preeminent.

During and after the war of 1914–18 the common war diseases were highly prevalent in eastern Europe and there was a vicious pestilence of typhus fever throughout Russia, Poland, and Serbia. Typhus did not spread in western Europe in these war years and the classic infections did not constitute the major sickness problems among fighting forces or civilians. The influenza pandemic overshadowed all others and the mortality from influenza and pneumonia recalled the ravages of the Black Death. Cerebrospinal meningitis reached new heights and tuberculosis and venereal disease increases caused grave concern.

In the United States Army influenza, venereal diseases, mumps, and measles were the most frequent causes of absence from duty and influenza, tuberculosis, measles, and cerebrospinal meningitis were leading primary causes of death. The Army in the United States had generally higher rates for admission and days lost from duty for the infectious diseases than did forces abroad. The United States Navy also suffered from the communicable diseases, with a notably higher incidence in the training camps than on shipboard.

In the present war, influenza and cerebrospinal meningitis are again showing increased incidence, both for the Continent and the British Isles. Typhus fever and dysentery have been active in eastern Europe.

The evacuation of school children did not result in the expected increase in the communicable diseases of childhood in England, and for the most part the health of the people has not been seriously affected. The hazards of infection have, however, been intensified by the crowding of the bomb shelters. Not only influenza and cerebrospinal meningitis but also paratyphoid fever and dysentery showed increases in 1940 in comparison with figures for 1939.

In the control of disease as in the fighting of battles the experience of the past can provide no rigid rules for present victory. Nevertheless an appreciation of former problems may facilitate their solution in later experience. The changed character of the leading infectious diseases of war, with relegation to the background of the scourges of

the past, may be considered a heartening reflection of the results of scientific research and control.

REFERENCES

(*1*) Annual report of the Surgeon General, U. S. Navy: (*a*) 1917; (*b*) 1918; (*c*) 1919; (*d*) 1920. Government Printing Office, Washington.
(*2*) Ayres, L. P.: The war with Germany. Government Printing Office, Washington, 1919.
(*3*) British Medical Journal, London: (*a*) **2**:300 (Sept. 1, 1917); (*b*) **1**:333–336 (Mar. 2, 1940); (*c*) **1**:629–631 (Apr. 13, 1940); (*d*) **1**:757–763 (May 11, 1940); (*e*) **2**:677–678 (Nov. 16, 1940).
(*4*) Britten, R. H., and Sydenstricker, E.: Mortality from tuberculosis in recent years. Pub. Health Rep., **37**:2843–2858 (Nov. 17, 1922). (Reprint 799.)
(*5*) Deutsche medizinische Wochenschrift, Berlin: Ausserordentliche Tagung des Deutschen Kongresses für Innere Medizin, Warschau, 1. u. 2. V., **42**:649–652 (May 25, 1916); 683–684 (June 1, 1916); 715–716 (June 8, 1916).
(*6*) Dorendorf and Kolle, W.: Klinische und bakteriologische Beobachtungen über Ruhr wahrend des Sommerfeldzuges einer Armee in Galizien und Russisch-Polen. Deutsch. med. Wchnschr., **42**:561–564 (May 11, 1916).
(*7*) Gaucher, P.-C.-E.: Les maladies vénériennes pendant la guerre à l'hôpital militaire Villemin et dans ses annexes. Bull. l'Acad. Méd., 3ᵉ série, **75**:352–360 (Mar. 28, 1916).
(*8*) Health, Australia: Australia and smallpox, **18**:4–8 (January 1940).
(*9*) Jordan, E. O.: Epidemic influenza. Am. Med. Assoc., Chicago, 1927.
(*10*) Lancet, London: (*a*) **2**:636–642 (Sept. 5, 1914); (*b*) **2**:562–564 (Sept. 23, 1916); (*c*) **2**:464–465 (Sept. 22, 1917); (*d*) **2**:1048–1049 (Nov. 11, 1939); (*e*) **2**:1082–1084 (Nov. 18, 1939); (*f*) **2**:1292 (Dec. 16, 1939); (*g*) **1**:440 (Mar. 2, 1940); (*h*) **1**:726–729 (Apr. 20, 1940); (*i*) **1**:762 (Apr. 20, 1940).
(*11*) League of Nations, Geneva: Epidemic Commission, (*a*) Report, 1921; (*b*) Second Annual Report, 1922.
(*12*) League of Nations: Epidemiological Intelligence, (*a*) Eastern Europe in 1921, No. 1, 1922; (*b*) Report of the European Health Conference, held at Warsaw, March 20–28, 1922; (*c*) Epidemics in Russia since 1914, Parts I and II, 1922.
(*13*) League of Nations: Weekly Epidemiological Record, (*a*) R. H. 707:410–413 (Sept. 14, 1939); (*b*) R. H. 720:543–547 (Dec. 14, 1939); (*c*) R. H. 735: 132–133 (Mar. 28, 1940); (*d*) R. H. 741:185–186 (May 9, 1940); (*e*) R. H. 761:327 (Sept. 26, 1940).
(*14*) Medical and surgical history of the War of the Rebellion (1861–65). Vol. I, Pt. III, Government Printing Office, Washington, 1888.
(*15*) Medical Department of the United States Army in the World War: (*a*) *6*. Sanitation. 1928; (*b*) *9*. Communicable and other diseases. 1928; (*c*) *15*. Pt. 2. Medical and Casualty Statistics. 1925. Government Printing Office, Washington.
(*16*) Prinzing, F.: Epidemics resulting from wars. Clarendon Press, Oxford, 1918.
(*17*) Rist, E.: (*a*) Acute contagious disease in the French Army. Am. J. Pub. Health, **7**:981–987 (December 1917); (*b*) The problem of pulmonary tuberculosis. Military Surg., **41**:659–668 (December 1917).
(*18*) Strong, R. P.: Typhus fever with particular reference to the Serbian epidemic. Harvard Univ. Press, Cambridge, 1920.
(*19*) Thomson, D. and R.: Annals of the Pickett-Thomson Research Laboratory. Influenza. Vol. 9, pts. 1 and 2. Williams and Wilkins Co., Baltimore, 1933–34.
(*20*) Waldman, A.: Wehrmedizinisches Vorwort zum 20. Gedenktag des Weltkriegsbeginns. Münch. med. Wchnschr., **81**:1157–1158 (Aug. 3, 1934).
(*21*) Wolff, G.: Tuberculosis mortality and industrialization. Pt. 1. Am. Rev. Tuberc., **42**:1–27 (July 1940).

See also: Index Medicus. War Supplement. A classified record on military medicine and surgery, 1914–1917. The Carnegie Institution of Washington, Washington, D. C.

CARBON DISULFIDE: ITS TOXICITY AND POTENTIAL DANGERS

Prepared by the Division of Industrial Hygiene, *National Institute of Health, United States Public Health Service*

Carbon disulfide is a toxic material which in high concentrations acts as a narcotic, whereas in low concentrations and with prolonged exposure it is a severe nerve poison.

Physical-Chemical Properties of Carbon Disulfide.

Pure carbon disulfide, CS_2, is a colorless liquid of a molecular weight of 76.13 and a specific gravity of 1.263 at $\frac{20^\circ}{4^\circ}$ C., if liquid, and of 2.63 against air. It has an unpleasant characteristic odor, melts at −111.6° C. and boils at 46.3° C. Its vapor pressure is 298 mm. at 20° C. and 433 mm. at 30° C. It is soluble in 100 parts of water to the extent of 0.14 parts at 49° C. and mixes freely with alcohol and ether. It is a good solvent for rubber, fats, and oils. It turns yellow upon standing under the influence of light. Its flash point is −25.5° to −20° C. and it has an extremely low ignition temperature of 120 to 156° C.; an air mixture containing 1.5 percent carbon disulfide flashes at 108° C. and even contact with moderately hot objects, such as steam pipes and electric bulbs, may cause ignition of the vapors. The lower explosive concentration limit is given as 1.06 volumes percent and the upper explosive concentration limit has been determined as 50 volumes percent at 6° C.

Maximal Permissible Concentration of Carbon Disulfide.

The maximal permissible concentration of carbon disulfide is at present accepted as 20 parts per million parts of air by volume,[1] corresponding to 0.062 milligrams per liter at 25° C. and 760 mm. Hg, for exposures not exceeding a total of 8 hours daily.

Sources of Exposure to Carbon Disulfide.

Exposure to carbon disulfide may exist in a number of industries.

In the *rayon industry* carbon disulfide vapors are developed, especially in the preparation of viscose and in the spinning and washing operations.

In the *rubber industry* the same hazard exists when carbon disulfide is used as a solvent for sulfur in cold vulcanization or as one of the solvents for rubber cement.

In the *fumigation industry* it is used as an insecticide.

In the *chemical industry* it is used as a solvent for fat, oils, and phosphorus. It is also used in the manufacture of carbon tetra-

[1] This figure for the maximal permissible concentration of carbon disulfide has been accepted and published by the American Standards Association in its Standard on Allowable Concentrations of Toxic Dusts and Gases—Z37.3-1941. Copies of this standard may be purchased from the American Standards Association, 29 West Thirty-ninth Street, New York, N. Y.

chloride, camphor, and, to a certain extent, in the manufacture of many products, such as waterproof cements.

Determination of Carbon Disulfide in Air.

For the determination of carbon disulfide in air, samples should be taken wherever there is a known or suspected source of carbon disulfide. They should be taken at the breathing level of the workers exposed, special emphasis being given to the locations nearest the source and those in the path of air currents carrying the gas. Samples should be taken in sufficient numbers and at sufficient intervals of time to allow the determination of the average and maximal exposures.

There are several methods for the determination of carbon disulfide in air. Some are based on the formation of ethyl xanthate in an alcoholic solution of potassium hydroxide. In these determinations a sufficient amount of air (at least 15 to 25 liters) is drawn through a solution of potassium hydroxide in absolute alcohol at a rate of 0.6 to 1.5 liters per minute after hydrogen sulfide and ammonia have been removed by lavage with cadmium chloride and with diluted sulfuric acid. The potassium ethyl xanthate formed in this way may be determined directly by iodometric methods as described by Matuszak (1932) (*1*) or by precipitation with copper sulfate and subsequent determination of the copper in the washed precipitate as used by Barthelemy (1939) (*2*) and by Frauenhof (1935) (*3*).

More recently the interaction of carbon disulfide with diethylamine and cupric acetate, resulting in the formation of cupric diethyldithiocarbamate, has been used for its determination in air by Viles (1940) (*4*) and has been suggested by the British Department of Scientific and Industrial Research (1939) (*5*). In the first procedure a certain volume of air is bubbled through a mixture of an alcoholic triethanolamine solution of diethylamine and cupric acetate and the carbon disulfide is determined by matching the yellow color, produced in this reagent by carbon disulfide, against standards prepared by the same procedure from alcoholic solutions of carbon disulfide of known concentrations. It appears that for this determination 160 to 360 cc. of air are sufficient and that it gives satisfactory results in a range of 10 to 80 parts per million. Data found with this method indicate that the method of sampling may have some effect on the results but that the analytical error is not more than about ± 1 percent for low concentrations, whereas with higher concentrations (80 parts per million) it may be distinctly greater.

Concentrations of Carbon Disulfide Determined under Various Conditions.

The concentrations found in various operations in which more or less severe toxic reactions have been observed range from 32 to 602 parts per million according to Constensoux and Heim (1910) (*6*),

Kranenburg and Kessener (1925) (*7*), Weise (1933) (*8*), Aïthoff (1905) (*9*), and Voltmer and Nuck (1933) (*10*).

Absorption and Excretion of Carbon Disulfide.

The absorption of carbon disulfide takes place mainly through the lungs. Sufficient quantities to cause toxic effects may be absorbed through the skin and, if taken orally, also from the gastro-intestinal tract. The excretion takes place mainly through the lungs but a small fraction seems to be excreted with the urine, the sweat, and the feces.

Determination of Carbon Disulfide in Blood and Urine.

For the determination of carbon disulfide in blood the same principles may be utilized as used for its determination in air. A micro method, based on the formation and titrimetric determination of potassium xanthogenate, was worked out by Harrower and Wiley (1937) (*11*) but this is tedious and time-consuming. Hunter (1940) (*12*) determined carbon disulfide in blood and urine by extracting these twice with sulfur-free petroleum ether and determining the CS_2 in these extracts with diethylamine and cupric acetate. The yellow color of cupric diethyldithiocarbamate is matched against standards prepared in the same way from alcoholic solutions of carbon disulfide of known concentrations.

Relation Between Concentrations of Carbon Disulfide in Air and Toxic Symptoms.

The effects of high concentrations of CS_2 in air are illustrated in table 1.

TABLE 1.—*Toxicity of carbon disulfide for man (according to Lehmann-Hess)*

[Flury and Zernik, 1931 (*14*)]

Symptoms	Concentrations	
	Milligrams per liter	Approximate parts per million by volume
Immediately fatal, or later with exposure to ½ to 1 hour	15	4,800
Dangerous to life in ½ to 1 hour (Hess)	10–12	3,200–3,850
Tolerated for ½ to 1 hour without immediate or late effects	3–5	960–1,600
Already effective with exposure for several hours (Hess)	1–1.2	320–390

Continued exposure to much lower concentrations will cause serious disturbances, as indicated by more recent observations in which toxic signs and symptoms observed in workers were correlated with concentrations of carbon disulfide as determined in such operations. Such data (*6, 7, 8, 9, 10*) indicate that concentrations of 30 to 40 parts per million of air may cause distinct subjective and objective disturbances with sufficient lengths of exposure.

Clinical Picture of Carbon Disulfide Poisoning.

Inhalation of high concentrations of carbon disulfide resulting in *acute poisoning* causes unconsciousness which may be preceded or followed by delirium and may end in death from respiratory failure. Less severe exposure may result in headache, giddiness, respiratory disturbances, precordial distress and gastro-intestinal disturbances. *Chronic carbon disulfide poisoning* is much more common than acute intoxications. It is characterized by a toxic effect on all nervous structures and also on the blood and other organs, and there may be transitory changes from the acute to the chronic form. Incipient chronic carbon disulfide poisoning is usually first characterized by subjective complaints such as fatigue, loss of memory, heaviness of the limbs and vertigo, which symptoms may rapidly disappear in fresh air. If the exposure, however, is continued or repeated, these symptoms may persist. In addition, there may be insomnia during the night and sleepiness during the day, weakness, loss of appetite, gastro-intestinal disturbances characterized by constipation or diarrhea, gastric pain, loss of weight, bad taste in the mouth, frequent urination, menstrual disturbances, primary increase and secondary decrease of libido. There may also be fainting spells, visual disturbances, staggering gait, various nervous disturbances such as reduction of corneal and palpebral reflexes, anesthesias and paresthesias, circumscribed paralysis resulting in atrophy of the corresponding muscle groups, tremors, and damage of the optic and acoustic nerves. Psychoses may develop, of manic or depressive character, and they may be of various intensities, but as a rule these are of transitory nature although permanent psychoses have been reported. In more severe chronic carbon disulfide poisoning, symptoms similar to Parkinsonism and multiple sclerosis have been reported, indicating the toxic effect on the corpus striatum, the globus pallidus, and the globus niger. The differentiation between these different conditions may offer considerable difficulties. The visual disturbances may vary considerably in intensity and character. In light cases they may be so moderate that they escape attention. Some patients complain of foggy vision. Others may suffer from color scotoma, reduction of the visual field, and, occasionally, from central scotoma. Most of these disturbances appear to be due to an affection of the optic nerve, the papilla, or the retina. Affections of the exterior muscles of the eyeball are exceptional.

The respiratory tract may show various degrees of irritation; there may be congestion of the lungs with bloody sputum, emphysema, and also chronic bronchitis.

The subjective signs from the gastro-intestinal tract mentioned above may become more severe with continued exposure and may develop into chronic gastritis and favor the formation of gastric and

duodenal ulcers. Spasms and dysfunction of the bladder have also been observed.

There is no evidence that carbon disulfide has a definite effect on the *circulatory apparatus* although palpitation of the heart, irregularities of the pulse, and bradycardia and tachycardia have been reported.

There is some evidence that continued exposure to carbon disulfide may affect the blood and possibly also the blood-forming organs, resulting in anemia characterized by reduction of the red blood cells and the hemoglobin, whereas the white blood cells may vary in their response. These occur, however, after nervous symptoms have developed.

Contact of carbon disulfide with the *skin* causes irritation and if maintained for a sufficient length of time it may result in severe pain, hyperemia, erythema, and blister formation which may have the tendency to form recidives, as demonstrated by Hueper (1936) (*15*) and by Oettel (1936) (*16*).

Table 2 gives information regarding symptoms observed in a group of 57 persons exposed to comparatively low concentrations of carbon disulfide (30 parts per million and more) as published by Voltmer and Nuck (1933) (*10*). This table appears to be especially instructive because it indicates which signs and symptoms should be looked for in periodical physical examinations of workers.

TABLE 2.—*Percentage incidence of toxic manifestations in a group of 57 persons exposed to moderate concentrations of carbon disulfide*

[Voltmer and Nuck, 1933 (*10*)]

	Percent		Percent
Subjectively and objectively normal	10.5	Nausea and vomiting and headache	38.6
Loss of appetite, gastro-intestinal disturbances, headache, loss of libido, excitement, depression	12.3	Forgetfulness and loss of memory	24.6
Polyuria	7	Slight visual disturbances	66.6
Loss of appetite	19.3	Sensory disturbances in different locations of the body	17.6
Intestinal irregularities (especially constipation)	33.3	Fatigue	[1] 100

[1] Practically.

Pathological Changes Observed in Carbon Disulfide Poisoning.

Pathological changes observed in acute carbon disulfide poisoning are characterized by hyperemia of the brain and other organs and multiple hemorrhages. In chronic forms with symptoms similar to Parkinson's disease, regressive changes and fatty degeneration of the liver, kidneys, and heart and, in addition, changes of the nervous system have been reported. The latter were more severe in the gray matter of the brain and in the pons where the pyramidal tracts showed severe degeneration. The corpus striatum and, to a lesser extent,

the globus pallidus, may show degenerative changes of the nerve cells and bundles with simultaneous proliferation of the neuroglial tissue (*18*). In cases of peripheral neuritis, the affected nerves and the muscles supplied by them may show various degrees of degeneration. Degenerative changes of the adrenals and of the pituitary have also been reported. The lungs may show emphysema and atelectases. The blood picture may show a reduction of the red blood cells with anisocytosis and poikilocytosis. As pointed out above, changes of the white cell picture are not constant. Some observers reported a monocytosis and eosinophilia (*19*).

Changes of Tolerance for Exposure to Carbon Disulfide.

As with other poisons, there appear to be considerable variations regarding the susceptibility to carbon disulfide, but it appears that there is no increase of the resistance against the toxic action; on the contrary, the poisoning may cause high susceptibility for further exposure.

Mechanism of Carbon Disulfide Poisoning.

Regarding the mechanism of carbon disulfide poisoning, it appears that carbon disulfide acts directly as a nerve poison and the question of whether or not it acts by some other mechanism has not been determined.

Prevention of Carbon Disulfide Poisoning.

In the prevention of carbon disulfide poisoning, proper instruction regarding its dangers of those who handle the material is very important.

Wherever possible, carbon disulfide should be handled in closed systems; if drained, it should be collected under water and it should be stored under water or under some inert gas which will not react chemically with carbon disulfide. Waste water containing carbon disulfide should not be drained into the sewer system.

When carbon disulfide is used as a solvent, the room should have adequate forced ventilation, the vapors should be removed at the site of their formation by proper exhaust ventilation and in order to prevent explosion this should be arranged in such a way that vapors do not come in contact with hot pipes, hot plates, or hot electric light bulbs. In operations where carbon disulfide is handled, the electric lights should be covered with vapor-proof globes, and any electrical or other devices which may produce sparks should be protected in such a way that any contact with carbon disulfide vapors is rendered impossible.

Regarding the allowable limits for the pollution of air with carbon disulfide, it is definitely known that continued exposure to 30 parts

per million is liable to cause toxic effects. Twenty parts per million has been accepted as the maximal permissible limit for continued exposure.

When rooms or enclosures containing carbon disulfide vapors have to be entered, air-supplied masks and safety belts should be worn. During the whole time, the workers should be watched by a supervisor familiar with the toxicity and potential dangers of carbon disulfide and with the first-aid treatment of accidents.

In order to prevent carbon disulfide poisoning, greatest personal hygiene is of paramount importance; any contact with the skin should be avoided. It should be emphasized that rubber gloves offer no adequate protection because carbon disulfide penetrates them readily and they interfere subsequently with the rapid evaporation from the skin. Serious injury of the subcutaneous tissue and the peripheral nerve endings may result from their use. Any soiled garments should be removed at once and any spills on the floor or tables should be removed and the rags used for this purpose should be discarded at once.

Workers exposed to carbon disulfide should have periodic examinations with special attention to subjective complaints. A thorough examination of the nervous system, including the examination of the eyeground, is essential. In order to prevent possible cumulative effects of carbon disulfide, periodic rotation to operations in fresh air of the workers exposed to this toxic agent appears advisable.

The treatment of carbon disulfide poisoning should consist in the first line in discontinuation of the exposure. In *acute poisoning* the patient should be transferred to fresh air and any soiled garments should be removed at once and, if necessary, the skin should be washed with large quantities of alcohol to remove the toxic material. The patient should be placed under the care of a physician as soon as possible. If the patient is in collapse, any chilling should be avoided by wrapping him in warm blankets and keeping him warm with hot water bottles with the necessary precautions to avoid direct contact with the skin. The application of analeptics such as caffeine sodium benzoate may become necessary. If the respiration is slow, irregular, or intermittent, administration of oxygen, alone or in combination with carbon dioxide, may become necessary. If the respiration has stopped but the heart is still beating, artificial respiration with the Shaefer prone pressure method should be instituted. Artificial respiration should be given only when indicated and then with proper precautions on account of the possibility of existing pulmonary hyperemia and emphysema.

In *chronic poisoning*, removal from further exposure is essential. Proper and adequate nutrition, fresh air, and other measures to improve

the physical condition are of great benefit and may be sufficient to overcome minor injuries. Severe cases of poisoning require symptomatic treatment.

REFERENCES

(*1*) Matuszak, M. P.: Iodometric determination of carbon disulfide. Ind. Eng. Chem., Anal. Ed., **4:** 98 (1932).
(*2*) Barthelemy, H. L.: Ten years' experience with industrial hygiene in connection with the manufacture of viscose rayon. J. Ind. Hyg. and Toxicol., **21:** 141 (1939).
(*3*) Frauenhof, H.: Determination of carbon disulfide and hydrogen sulfide in viscose rayon plant atmosphere. Kunstseide, **17:** 344 (1935).
(*4*) Viles, F. J.: Field determinations of carbon disulfide in air. J. Ind. Hyg. and Toxicol., **22:** 188 (1940).
(*5*) Department of Scientific and Industrial Research: Methods for the detection of toxic gases in industry. Leaflet No. 6, Carbon disulfidé. His Majesty's Stationery Office, London, 1939.
(*6*) Constensoux, M. G., and Heim, M. F.: Relative frequency of nervous signs in chronic carbon disulfide poisoning. Second Congress on Industrial Diseases, Brussels, 1910.
(*7*) Kranenburg, W. R. H., and Kessener, H.: Hydrogen sulfide and carbon disulfide poisonings. Zentr. Gewerbehyg., **12:** 348 (1925).
(*8*) Weise, W.: Gastro-intestinal disturbances from chronic carbon disulfide and hydrogen sulfide inhalation. Arch. Gewerbepath. Gewerbehyg., **4:** 219 (1933).
(*9*) Aïthoff: Contribution to the effects of carbon disulfide. Thèse de Paris, 1905.
(*10*) Voltmer and Nuck: Studies on the question of health injuries of workers from vulcanization of rubber goods. Reichsarbeitsblatt, **3:** 2, 21 (1933).
(*11*) Harrower, J. R., and Wiley, F. H.: The determination of carbon disulfide in blood. J. Ind. Hyg. and Toxicol., **19:** 486 (1937).
(*12*) Hunter, A. W.: The determination of carbon disulfide in blood and urine. J. Ind. Hyg. and Toxicol., **22:** 231 (1940).
(*13*) Lehmann, K. B.: Experimental studies on the influence of technically and hygienically important gases and vapors on the organism. VII. Carbon disulfide and sulfur chloride. Arch. Hyg., **20:** 26 (1894).
(*14*) Flury, F., and Zernik, F.: Schädliche Gase. Julius Springer, Berlin, 1931, p. 297 ff.
(*15*) Hueper, W. C.: Etiologic studies on the formation of skin blisters in viscose workers. J. Ind. Hyg. and Toxicol., **18:** 432 (1936).
(*16*) Oettel, H.: Effect of organic liquids on the skin. Arch. exp. Path. Pharmakol., **183:** 674 (1936).
(*17*) Ranelletti, A.: Occupational CS_2 poisoning. Arch. Gewerbepath. Gewerbehyg., **2:** 664 (1931).
(*18*) Abe, M.: Contribution to the pathological anatomy of chronic carbon disulfide poisoning. Japan, J. Med. Sci. VIII. Intern. Med., **3:** 1 (1933).
(*19*) Bignami, G.: Blood changes in CS_2 poisoning. Boll. soc. med.-chir. Pavia., **37:** 745 (1925).

EXPERIMENTAL POLIOMYELITIS [1]

The Use of a Variety of Laboratory Techniques in Efforts to Establish Seven Strains of Poliomyelitis Virus in the Cotton Rat

By S. D. Kramer, M. D., and W. N. Mack, with the assistance of A. T. Himes

In view of the importance to poliomyelitis research of Armstrong's successful transmission of one strain of poliomyelitis virus to the eastern cotton rat and white mouse (*1*, *2*), we undertook a study to

[1] From the Bureau of Laboratories of the Michigan Department of Health, Lansing, Mich. Aided by a grant from the National Foundation for Infantile Paralysis, Inc.

determine the regularity with which other strains of the virus might be established in these animals. The procedures employed in these attempts are listed and described below.

Seven strains of virus and a total of 257 eastern cotton rats were used in this investigation. Of these seven strains one was our own V. M. old monkey laboratory strain and the remaining six were recently isolated strains, three in our own laboratory from the feces of contacts and three isolated by Dr. John Kessel from post-mortem material. In addition, one series of rats was inoculated with infective fecal material.[2] (See table 1.)

That infection of the cotton rat is not readily accomplished by the usual laboratory procedures is evidenced from Armstrong's long efforts, from Toomey's first negative experience with nine strains of virus (*6*) in which 68 rats were used, from Hammon's similarly negative results (*7*), and from our own failure to establish any strain successfully in these animals. In spite of our failure we have, in the course of our investigations, encountered a number of suggestive symptoms and histologic changes in rats which encouraged us to continue these investigations and prompted us to report these negative findings.

In two recent reports (*8, 9*) of the successful transmission of monkey poliomyelitis to cotton rats and white mice, the authors emphasized certain technical procedures which they employed and believed instrumental in obtaining positive results. In our own efforts we included most of the technical procedures that have in our experience or that of others proved useful in connection with poliomyelitis and other viruses. These are (1) the method of rapid passage, (2) mechanical trauma and trauma by starch, (3) reinforcing reinoculations, (4) use of the "spreading factor of Duran-Reynals," (5) hyperpyrexia, (6) chilling, and (7) the use of young, immature animals.

STRAINS

Table 1 lists the strains of virus employed, their source, and the number of animals inoculated with each. As can be seen from the table, strain D. G. received the most attention. This strain had given a rapid, prostrating paralysis in its first monkey passage.

[2] This stool was proved positive by successful monkey inoculation and illustrates the survival of the virus in the feces for over 6 months at ice-box temperature (*4*). It was concentrated by the usual procedure (*5*).

TABLE 1.—*Strains of virus employed*

Strain	Source of strain	Monkey passages	Number of rats which received				Total rats
			Fresh monkey cord	Glycerated monkey cord	Fecal material	Rat material	
D. G	Feces	5, 6, 7, 8, 9	3	41		137	181
B. M	Feces	4		2		4	6
S. M	Feces	1, 2	6	10		5	21
S. M. (feces) [1]					4	13	17
Stock	Old monkey passage			1		3	4
Kessel Mc	C. N. S	2		2			2
Kessel K	C. N. S	2		2		20	22
Kessel Sch	C. N. S	5, 6	2	2			4
Total			11	60	4	182	257

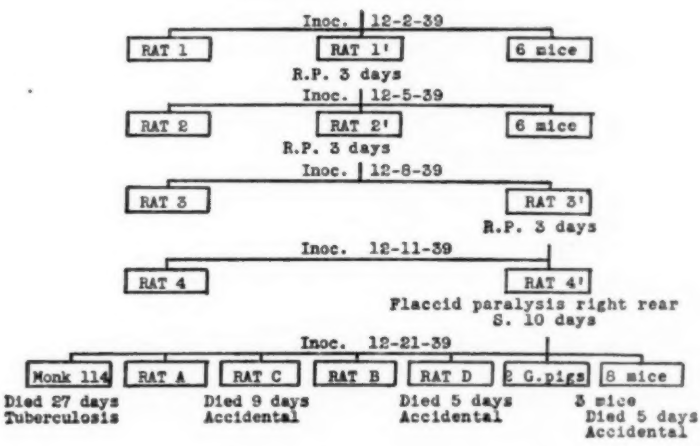
[1] See footnote 2.

TECHNIQUES EMPLOYED

1. Our method for rapid passage is illustrated in charts I and II. Two or more animals were inoculated, one or more of which were

CHART I

POOLED D.G. MONKEY CORDS*



* Monkey cords pooled: 22,28,61,67,102

sacrificed after 72 hours and passage made to a second series of two or more animals, one or more of which were sacrificed after 72 hours. This was repeated until a fourth passage was made. This procedure made it possible for us to observe both the parallel animals not sacrificed and the effect of rapid passage. In this, as in every other special procedure, whenever a surviving animal presented suggestive symptoms, it was sacrificed and 10 percent suspensions of the cord and brain were promptly inoculated into other rats.

2. Following the method successfully used by Sawyer in establishing the yellow fever virus in mice (*10*) we inoculated the rats with 0.06 to

0.2 cc. of a 2 percent soluble starch solution into one hemisphere and 5 to 20 minutes later we inoculated the 10 percent virus suspension in the opposite hemisphere. From time to time in the course of our inoculations we produced intentional cortical trauma by rotating the needle after insertion into the brain substance.

3. Following Flexner's suggestion (*11*) that subinfective doses could be made infective by periodic reinoculations, we reinoculated a large number of rats (see chart II) at weekly intervals for 1 or 2 successive weeks. In two instances animals were reinoculated with the concentrated S. M. fecal material.

4. The "spreading factor principle of Duran-Reynals" (*12*) was used in a small group of four animals. A 10 percent orchitic extract in saline prepared from untraumatized testes of a normal rabbit was used as diluent in preparing the 10 percent virus suspension.

5. Four animals were exposed to the radiations of a G. E. inductotherm short wave machine for 10 to 18 minutes until the animals' temperatures were elevated to from 105° to 109.4° F. These animals were immediately inoculated with the virus suspensions.

6. Seven rats, four of which had received preliminary chilling in the ice chest (3°–4° C.) for 24 hours, were inoculated and then kept at ice-box temperature. Two animals survived 52 days under these conditions. A number of the animals died in 3 days; two animals became ill with indefinite symptoms, were sacrificed, and passage was made into normal rats.

7. Six immature animals ranging in age from 1 to 3 weeks were inoculated with 10 percent virus suspensions.

Table 2 indicates the number of animals used for each special procedure and strain.

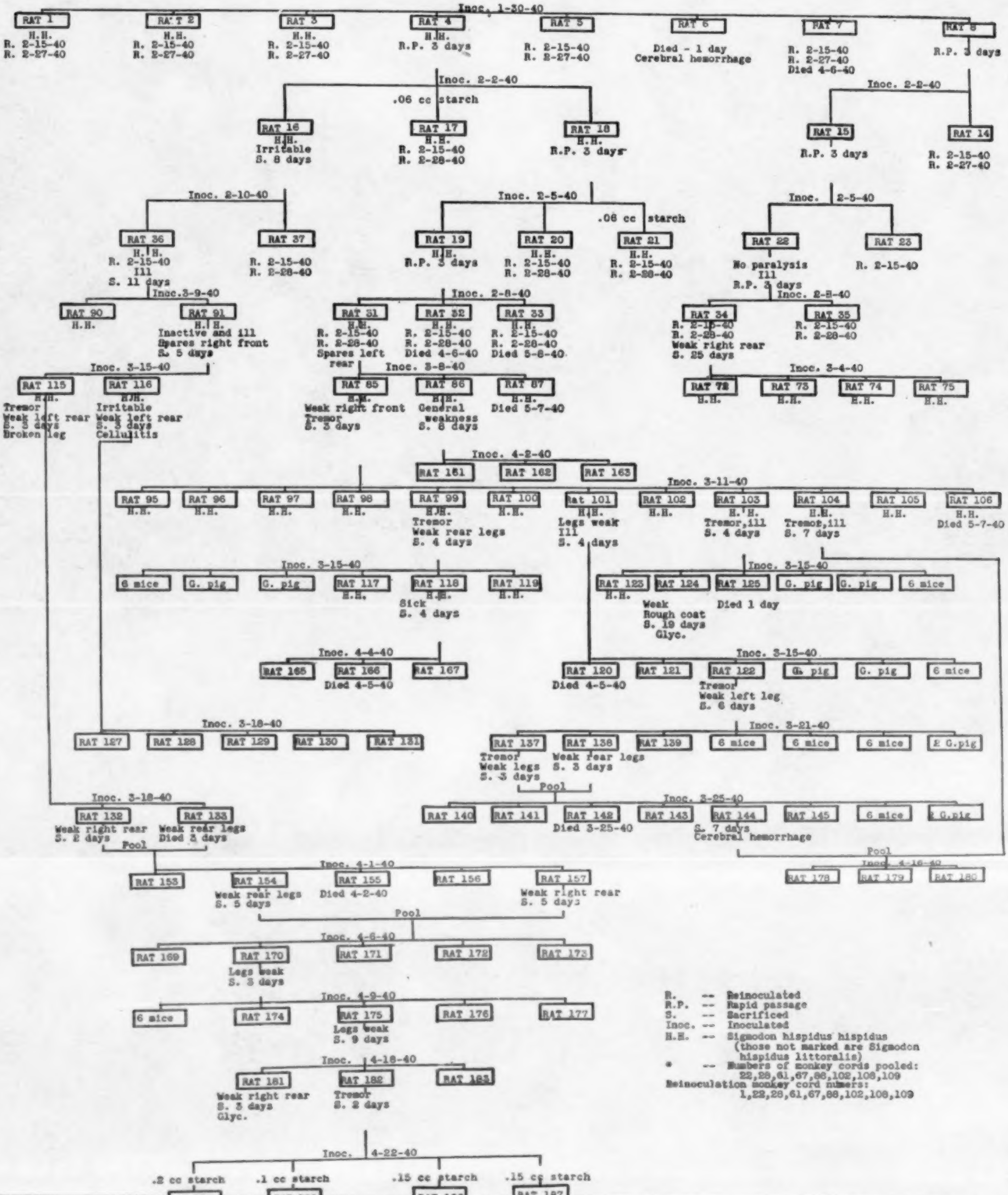
TABLE 2.—*Number of animals used*

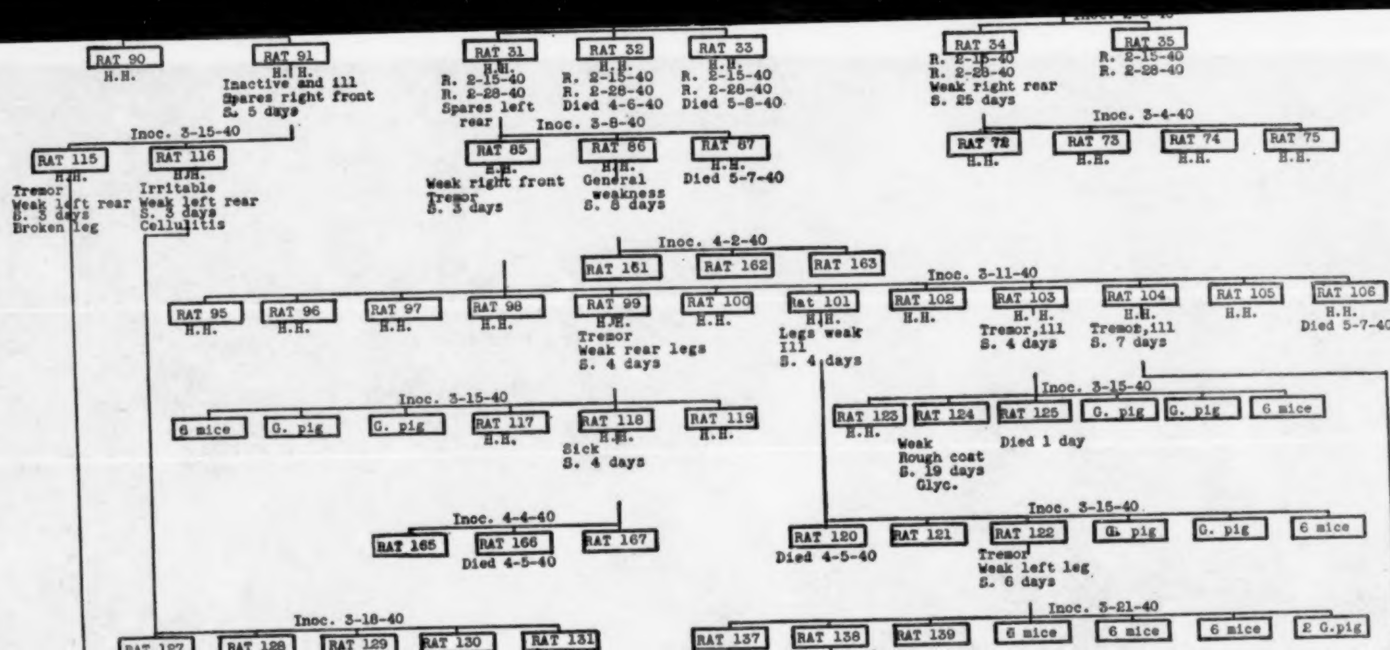
Strain	Rapid passage	Preliminary starch inoculation	Reinoculations		Orchitic extract	Hyperpyrexia	Chilling	Immature animals	Total rats
			Number of rats	Number of reinoculations					
D. G.	29	7	47	89	4	4	3	6	100
B. M.	6								6
S. M.							4		4
S. M. (feces)			2	2					2
Stock	2								2
Total	37	7	49		4	4	7	6	114

The remainder of the 257 rats were inoculated following Armstrong's procedures or were used for passage of material from suspicious animals. In addition, 187 Swiss strain mice, 12 guinea pigs, and 3 monkeys were inoculated in the various experiments.

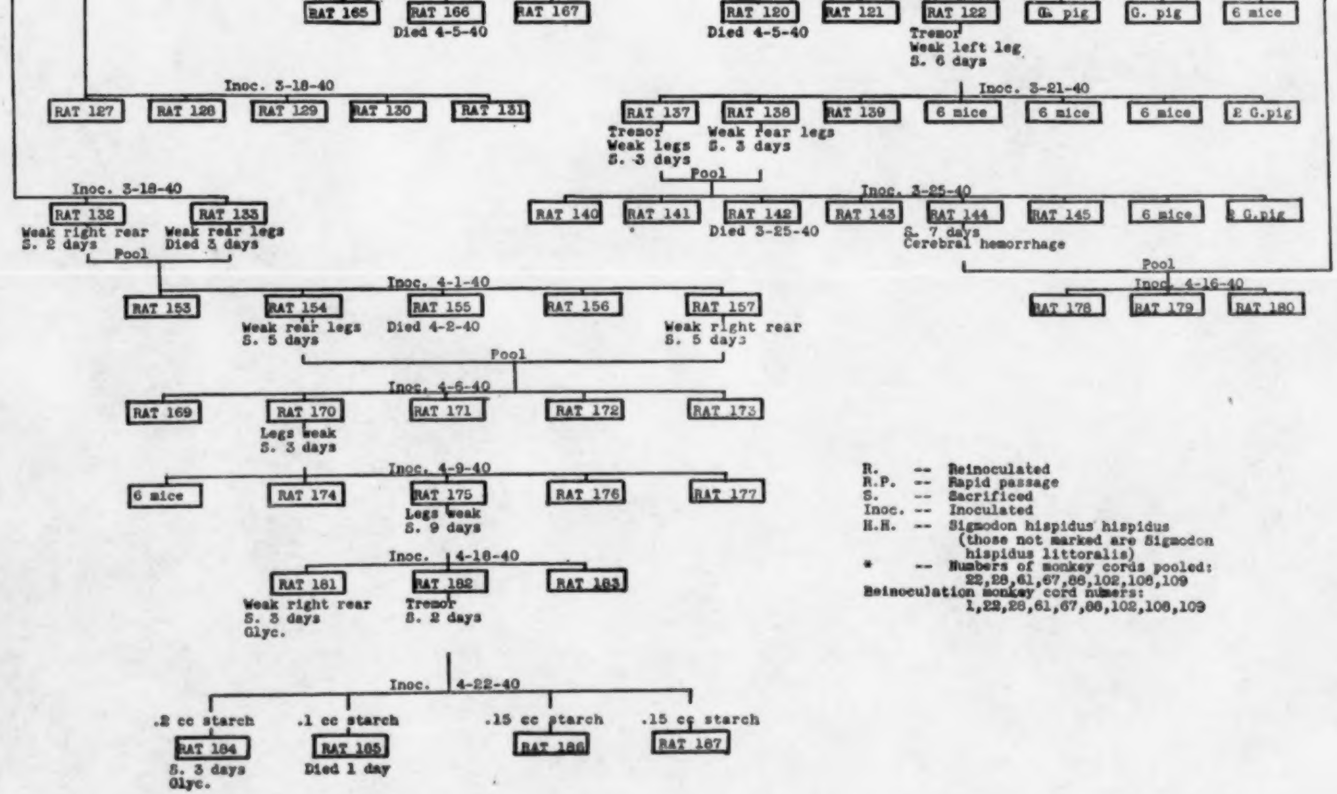
CHART II

POOLED MONKEY CORDS D.G. STRAIN*

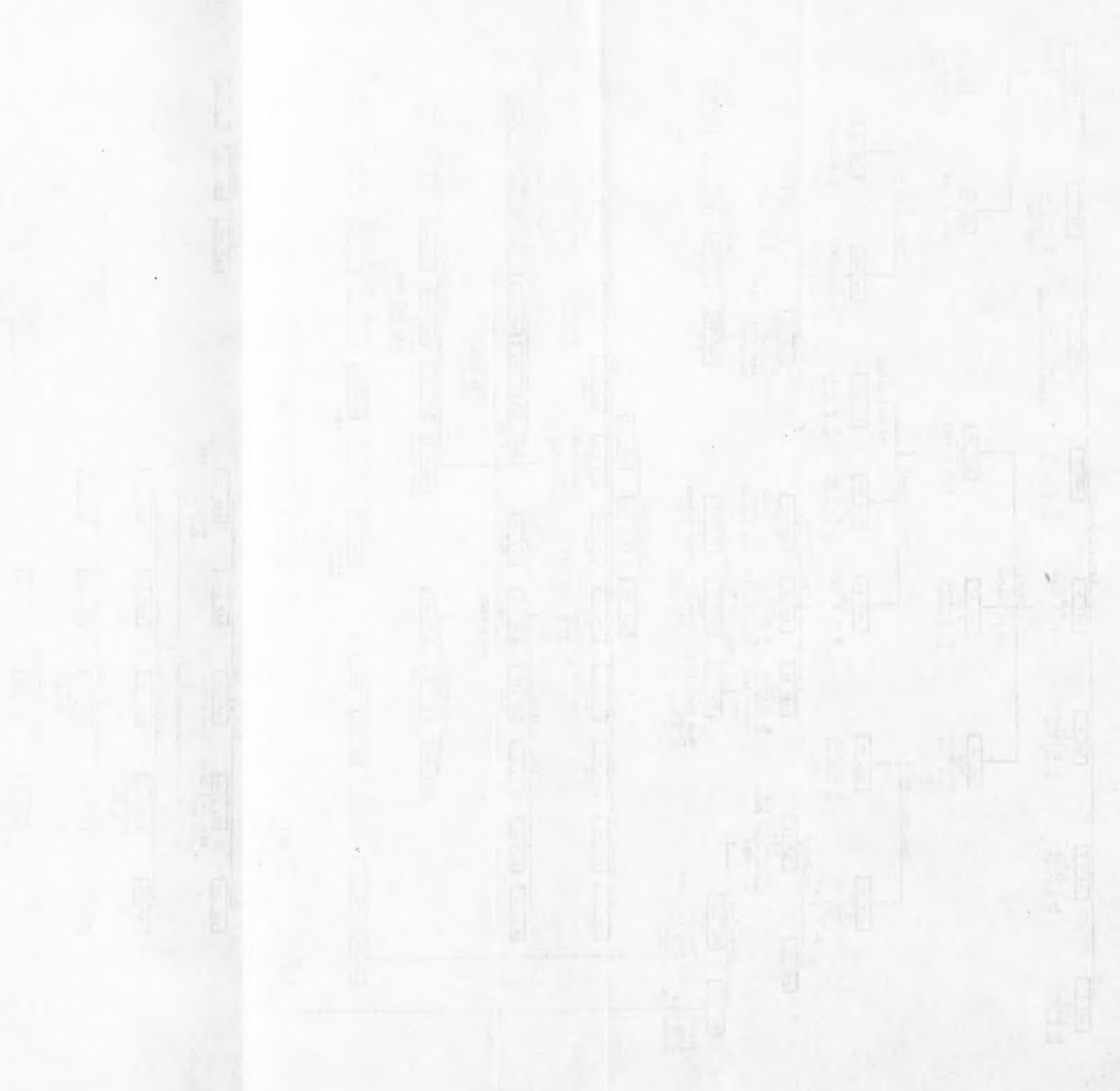




RAT 90
H.H.
RAT 91
H.H.
Inactive and ill
Spares right front
S. 5 days
RAT 31
H.H.
R. 2-15-40
R. 2-28-40
Spares left rear
RAT 32
H.H.
R. 2-15-40
R. 2-28-40
Died 4-6-40
RAT 33
H.H.
R. 2-15-40
R. 2-28-40
Died 5-8-40
RAT 34
R. 2-15-40
R. 2-28-40
Weak right rear
S. 25 days
RAT 35
R. 2-15-40
R. 2-28-40
Inoc. 3-15-40
RAT 115
H.H.
Tremor
Weak left rear
S. 3 days
Broken leg
RAT 116
H.H.
Irritable
Weak left rear
S. 3 days
Cellulitis
Inoc. 3-8-40
RAT 85
H.H.
Weak right front
Tremor
S. 3 days
RAT 86
H.H.
General weakness
S. 8 days
RAT 87
H.H.
Died 5-7-40
Inoc. 3-4-40
RAT 72
H.H.
RAT 73
H.H.
RAT 74
H.H.
RAT 75
H.H.
Inoc. 4-2-40
RAT 161
RAT 162
RAT 163
Inoc. 3-11-40
RAT 95
H.H.
RAT 96
H.H.
RAT 97
H.H.
RAT 98
H.H.
RAT 99
H.H.
Tremor
Weak rear legs
S. 4 days
RAT 100
H.H.
Rat 101
H.H.
Legs weak
Ill
S. 4 days
RAT 102
H.H.
RAT 103
H.H.
Tremor,ill
S. 4 days
RAT 104
H.H.
Tremor,ill
S. 7 days
RAT 105
H.H.
RAT 106
H.H.
Died 5-7-40
Inoc. 3-15-40
6 mice
G. pig
G. pig
RAT 117
H.H.
RAT 118
H.H.
Sick
S. 4 days
RAT 119
H.H.
Inoc. 3-15-40
RAT 123
H.H.
RAT 124
Weak
Rough coat
S. 19 days
Glyc.
RAT 125
Died 1 day
G. pig
G. pig
6 mice
Inoc. 4-4-40
RAT 165
RAT 166
Died 4-5-40
RAT 167
Inoc. 3-15-40
RAT 120
Died 4-5-40
RAT 121
RAT 122
Tremor
Weak left leg
S. 6 days
G. pig
G. pig
6 mice
Inoc. 3-18-40
RAT 127
RAT 128
RAT 129
RAT 130
RAT 131
Inoc. 3-21-40
RAT 137
RAT 138
RAT 139
6 mice
6 mice
6 mice
2 G.pig



292182°—41 (Face p. 584)



In every instance when an animal was sacrificed for passage, fragments were taken from the central nervous system axis and occasionally from other systems and preserved in formalin or other preservative for histologic study.

The methods of inoculation were essentially those outlined by Armstrong. From 0.1 to 0.3 cc. of a 10 percent suspension of monkey or rat material was inoculated intracerebrally. The animals appeared to stand these larger amounts quite well. In addition to the intracerebral inoculations most of the animals received supplementary inoculations intraperitoneally (0.5–1.0 cc.), subcutaneously (0.2–0.5 cc.), or intranasally (0.03 cc.). For the intranasal instillations the sediment from the centrifuged virus suspension was resuspended in a little saline. At the outset of this investigation the entire rat brain and cord were ground to make the 10 percent suspension and used as inoculum. After some experience with the Armstrong strain where we found that essentially all of the virus was located in the cord and medulla, this portion of the cerebrospinal axis alone was used in the preparation of suspensions.

RESULTS

We were unsuccessful in establishing any of the seven strains of virus in the eastern cotton rat and the white mouse. At the very outset and on a number of later occasions we had reason to believe that our efforts would be fruitful, and it is largely on the basis of these suggestive leads that we are led to report our negative results. Rat 4′ (chart I), the fourth animal in one of the rapid passage series, developed a flaccid paralysis of the right rear extremity 10 days after inoculation. This animal was sacrificed, brain and cord emulsified, and a 10 percent suspension inoculated into four rats. (Shortage of animals at this time limited us to this number.) Two of the four rats were accidentally killed. The surviving two rats remained symptom-free. Monkey 114 inoculated intracerebrally and intraperitoneally with material from this animal failed to present symptoms and died of tuberculosis on the twenty-seventh day after inoculation. Rat 4′ was the only animal in all of our attempts that developed a frank flaccid paralysis; nevertheless, the paralysis was typical, and no other cause could be found. Histologic examination of two levels of the cord showed it to be edemic. The motor cells were irregular in size and shape and exhibited degenerative changes ranging from the loss of nuclear material to almost complete degeneration. Inflammatory reaction was only slight and only occasional polymorphonuclear cells were observed. No instance of spontaneous paralysis has been seen in almost 1,000 rats which we have observed during the past 14 months.

From time to time in the course of the investigation other animals appeared to favor one or more extremities and these were promptly sacrificed and material passed on to other rats. However, none of the subsequently injected animals, even when the rapid passage or other procedures were employed, developed typical paralysis.

In one group of rats beginning with the fourth animal of a rapid passage series (rat 31) and which had also received two fortifying inoculations a peculiar syndrome was observed (see chart II). This animal developed some weakness of the left rear leg 8 days after the last reinoculation. It was sacrificed and 10 percent brain and cord suspension inoculated into three rats, one of which, on the third day, developed a tremor and a weakness of the right front leg. This animal was sacrificed and the brain and cord inoculated into 12 rats, four of which developed a tremor, general weakness, and spared one or more extremities 3 days following inoculation. Nearly all of the remaining eight rats presented similar symptoms to a varying degree from which they recovered after a few days. The four animals with symptoms were sacrificed and material from each of them was inoculated into three rats. As indicated in the chart these symptoms continued for four passages but none of the animals developed flaccid paralysis characteristic of the Armstrong strain or similar to that obtained in our own first rapid passage series (rat 4′). Histologic examination of sections of the cords of animals sacrificed in this series (rats 31, 85, 86, 101, 103, 104, 118, 120, 122, 124, 137, 138, 144) (see chart II) has presented suggestive changes. The outstanding features have been edema, congestion, diffuse microscopic hemorrhages, and changes in the anterior horn cells from poor staining quality and smudging to loss of nuclear substance, pyknosis, and disappearance. Infiltration has not been a prominent part of the histologic picture. However, in our experience, extensive infiltration has not been an outstanding feature in the cord sections of rats succumbing to the Armstrong strain.

DISCUSSION

Jungeblut (*8*), in a recent description of a murine strain which he believes is a successful infection of the cotton rat with the poliomyelitis virus, emphasizes the possible importance of rapid passage, particularly of animals presenting symptoms or dying following inoculation. Although this procedure was repeatedly employed and on at least one occasion did appear to result in successful infection, it would seem that this method cannot be uniformly employed with success. In this study we did not employ enteric toxin as diluent, as recommended by Toomey (*9*) in his recent report of successful infection of the cotton rat and white mouse. In our experience as well as in the more recent

experience reported by Hammon (7) the "spreading factor of Duran-Reynals" proved ineffective.

After what is now a rather extensive experience with the Armstrong strain of poliomyelitis in rats and mice, we are convinced that this strain of virus is indistinguishable from monkey poliomyelitis produced with other strains in that animal. And, although we have been unable to find any single technical procedure which will readily yield successful infection in rats, the single instance of frank, flaccid paralysis and the number of instances of what appeared to be partial paralysis offer sufficient encouragement for the continuation of these investigations.

ACKNOWLEDGMENT

The authors gratefully acknowledge the helpful cooperation of Dr. H. E. Cope, pathologist of the Michigan Department of Health, in the preparation and study of the pathological material.

REFERENCES

(*1*) Armstrong, C.: Pub. Health Rep., **54**: 1719–1721 (1939).
(*2*) Armstrong, C.: Pub. Health Rep., **54**: 2302–2305 (1939).
(*3*) Kramer, S. D., Gilliam, A. G., and Molner, J. G.: Pub. Health Rep., **54**: 1914–1922 (1939).
(*4*) Kramer, S. D.: J. of Mich. State Med. Soc., October 1940.
(*5*) Kramer, S. D., Sobel, A. E., Grossman, L. H., and Hoskwith, B.: J. Exp. Med., **64**: 173–182 (1936).
Kramer, S. D., Hoskwith, B., and Grossman, L. H.: J. Exp. Med., **69**: 49–67 (1939).
(*6*) Toomey, J. A., and Takacs, W. S.: Proc. Soc. Exp. Biol. and Med., **43**: 536–537 (1940).
(*7*) Hammon, W. M.: Proc. Soc. Exp. Biol. and Med., **45**: 124–126 (1940).
(*8*) Jungeblut, C. W., and Sanders, M.: J. Exp. Med., **72**: 407–436 (1940).
(*9*) Toomey, J. A., and Takacs, W. S.: Proc. Soc. Exp. Biol. and Med., **45**: 364–366 (1940).
(*10*) Sawyer, W. A., and Lloyd, W.: J. Exp. Med., **54**: 533–555 (1931).
(*11*) Flexner, S.: Science, **74**: 520 (1931).
(*12*) Duran-Reynals, F.: J. Exp. Med., **50**: 327 (1929).

THREE NEW SPECIES OF *ORNITHODOROS* (ACARINA: IXODOIDEA) [1]

By R. A. Cooley, *Entomologist*, and Glen M. Kohls, *Assistant Entomologist, United States Public Health Service*

A new *Ornithodoros* found on a white-footed mouse and two new ticks of the same genus found on bats and in bat-inhabited caves and mines are here described. The species from *Peromyscus* was collected in southeastern Utah by field workers of the United States Public Health Service Plague Laboratory at San Francisco. The two species from bats were found in California and Arizona and additional specimens of one of them were obtained in Texas and Oklahoma.

[1] From the Rocky Mountain Laboratory, Hamilton, Mont., Division of Infectious Diseases, National Institute of Health.

Ornithodoros eremicus n. sp.

Body.—(Nymph.) Short oval (approaching circular), broadly rounded on both ends; tips of the mouth parts visible from above. Length 1.44 mm.; width 1.11 mm.

Mammillae.—Numerous, small, and of about equal size in median and peripheral areas on both dorsal and ventral surfaces. Individual mammillae only a little elevated, flattened, with their surfaces smooth and shining. A few short hairs present on the dorsum; those on the anterior margin and above the anterior legs are longer.

Discs.—Little in evidence, small, and slightly depressed; absent on the venter.

Legs.—Moderate in length and size. Surface smooth. Hairs few in number. Subapical dorsal protuberances present on tarsi I, II, and III, absent on tarsus IV. Dorsal humps moderate; three on

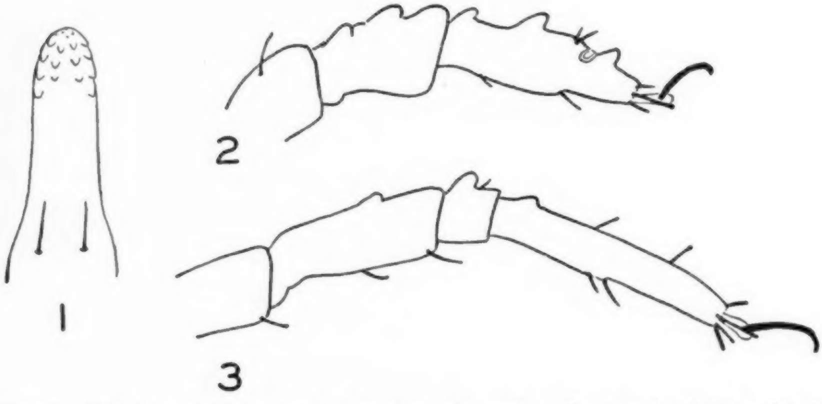


FIGURE 1.—*Ornithodoros eremicus* n. sp. 1. Hypostome of nymph. 2. Tarsus and metatarsus of leg I of nymph. 3. Tarsus and metatarsus of leg IV of nymph.

tarsus I, two on II, two on III, none on IV. Metatarsi I, II, and III each with three dorsal humps; metatarsus IV, with two. Length of tarsus I, 0.3 mm.; metatarsus, 0.18 mm. Length of tarsus IV, 0.42 mm.; metatarsus, 0.285 mm.

Coxae.—All coxae contiguous.

Hood, camerostome, and movable cheeks.—Absent.

Hypostome.—Moderate in length, sides parallel, apex rounded. Denticles arranged 2/2 with 2 or 3 teeth in each file, placed near the distal end. Length about 0.12 mm. (Description and drawing made with the hypostome *in situ*, not mounted.)

Folds.—Coxal and supracoxal folds present.

Grooves.—Dorso-ventral and preanal grooves present. Median postanal and transverse postanal grooves absent.

Eyes.—Absent.

Anus.—In a nearly circular frame.

This tick is described from a single small nymph received from Surgeon L. B. Byington, officer in charge, San Francisco Plague Laboratory. It is notable that, although the specimen is so small, dorsal humps on the tarsi are well developed. Since in all known American species the dorsal humps are poorly developed in the early nymphal stages and become progressively larger in the successive stages, it appears likely either that the adult would be very small, or, if of average size, the humps would be unusually prominent.

This new tick resembles the African species *O. savignyi* (Audouin).

Holotype.—A. P. 16314 from *Peromyscus maniculatus*, August 24, 1939, near Bluff, San Juan County, Utah, deposited in the collection of the Rocky Mountain Laboratory.

Ornithodoros stageri n. sp.

Body.—Oval, wider behind, and faintly pointed in front; somewhat flattened but with no change in structural pattern of the mammillae at the margins; tips of the palpi visible from above. Size of the holotype female 4.5 mm.×2.64 mm.; allotype male 3.84 mm×2.40 mm. Smallest and largest specimens, females, 3.3 mm.×2.4 mm. to 5.3 mm.×3.4 mm.; males, 3.3 mm.×2.3 mm. to 4.10 mm.×2.75 mm.

Mammillae.—Relatively large, few in number, and not crowded as in some species; irregular in shape, their tops smooth, convex, often with radial ridges on their bases. A few have a single, faint pit on the top which may or may not have a short, fine hair.

Discs.—Distinct, large, mostly circular; in depressed areas, but with elevated margins; present also on the venter where they are in lineal arrangement in or near the preanal, transverse postanal, and median postanal grooves.

Legs.—Moderate in length and size, with the surface smooth or very finely granulated, with barbed hairs moderate in number and in length. Dorsal humps and subapical protuberances absent. Length of female tarsus I, 0.66 mm., metatarsus 0.48 mm.; tarsus IV, 0.84 mm., metatarsus 0.75 mm. Length of male tarsus I, 0.45 mm., metatarsus 0.3 mm.; tarsus IV, 0.63 mm., metatarsus 0.54 mm.

Coxae.—Coxae I and II a little separated; all others contiguous.

Hood.—Negligible or absent.

Camerostome.—Faintly indicated.

Movable cheeks.—Present in the female; small, and somewhat variable in shape. Usually oval and attached at one side of the broad end. Absent in the male.

Capitulum.—Basis about as wide as long; surface irregular with transverse wrinkles. With two or three barbed hairs on each side on the lateral walls and a pair of spines back of the posthypostomal hairs,

which are more separated and smaller. Palpal article 1 with a thin flange projecting over the base of the hypostome.

Hypostome.—In both sexes the hypostome measures about 0.22 mm. and is notched, but there are structural differences in the

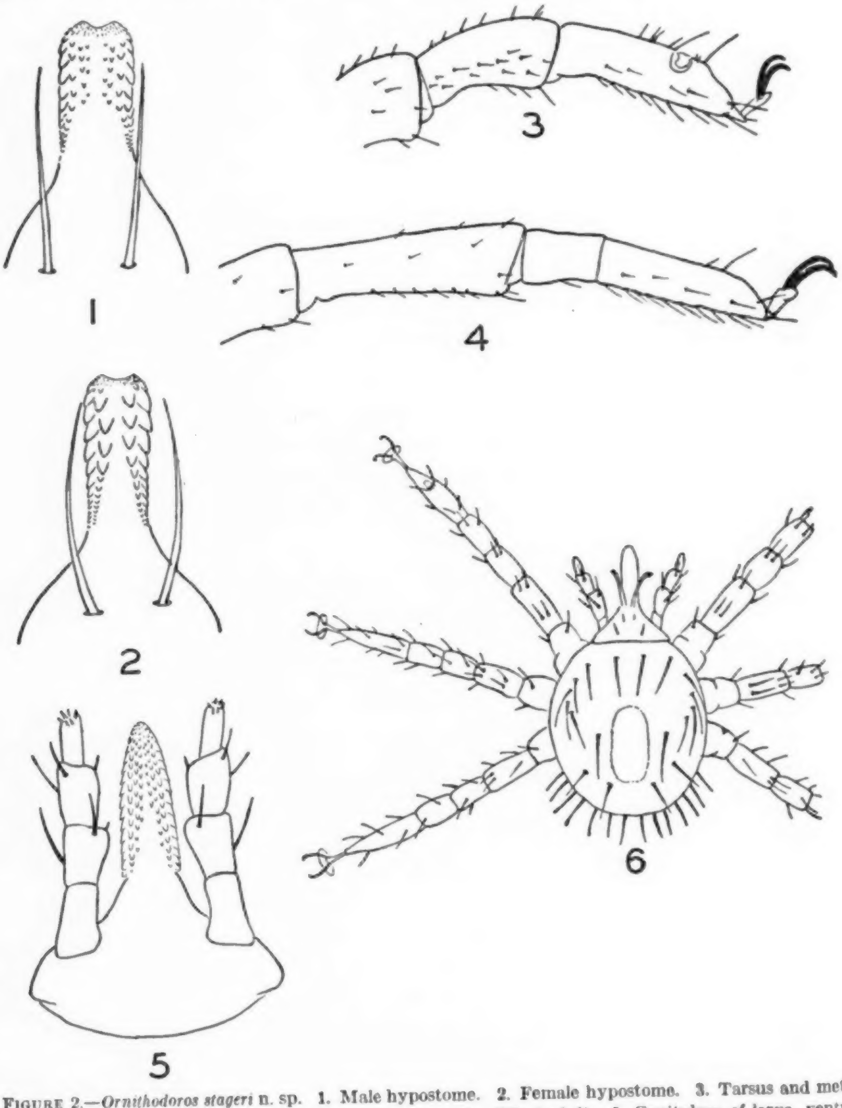


FIGURE 2.—*Ornithodoros stageri* n. sp. 1. Male hypostome. 2. Female hypostome. 3. Tarsus and metatarsus of leg I of adult. 4. Tarsus and metatarsus of leg IV of adult. 5. Capitulum of larva, ventral view. 6. Larva, dorsal view.

sexes. In the female the principal denticles, arranged 2/2, have the four files approximately equidistant, while in the male the hypostome is proportionately wider, has the denticles smaller with the two principal files separated by two short files of smaller denticles on each side of the median line.

Folds.—Coxal and supracoxal folds present.

Grooves.—Dorso-ventral groove absent; preanal, transverse postanal, and median postanal grooves present.

Sexual opening.—Placed at the level of the interval between coxae I and II.

Eyes.—Absent.

Anus.—In an elliptical pattern.

LARVA

Short oval, moderate in size. Length including hypostome 0.765 mm.; width 0.48 mm. With an oval area on the dorsum, which is shiny and with faint pits (visible with reflected light in unmounted specimens). Legs about as long as the length of the body, and with claws. Capitulum terminal and visible from above; basis broad. Hypostome lacking the distinct conical base found in some species (fig. 3–1); sides a little converging anteriorly, bluntly pointed apically. Denticles apically 4/4, then 3/3, and finally 2/2 at the base; those of the lateral files large, and those of the median files very small. Length of hypostome about 0.2 mm.

Holotype.—Female from A. P. 17868.

Allotype.—Male from A. P. 17868.

Paratypes.—29 females, 29 males, from A. P. 17868.

Holotype, allotype, and paratypes deposited in the collections of the Rocky Mountain Laboratory, except that single males and females have been deposited as follows: United States National Museum, Washington, D. C.; Zoological Division, Bureau of Animal Industry, Department of Agriculture, Washington, D. C.; Los Angeles County Museum, Los Angeles, Calif.; Division of Entomology and Parasitology, University of California, Berkeley, Calif.; Museum of Comparative Zoology, Harvard University, Cambridge, Mass.; Department of Entomology, Cornell University, Ithaca, N. Y.; Division of Entomology and Economic Zoology, University of Minnesota, Minneapolis, Minn.; and Department of Entomology, Oklahoma Agricultural and Mechanical College, Stillwater, Okla.

This tick is named in honor of Mr. Kenneth E. Stager, Los Angeles, Calif.

Four collections of this species from Texas, Oklahoma, Arizona, and California, respectively, have been made as follows: A. P. 17798, Ney Cave, August 5, 1939, 20 miles north of Hondo, Tex., one male, one female, one nymph (Kenneth E. Stager); A. P. 17859, on bat guano in Senator Mine, May 21, 1940, 21 miles northeast of Yuma, Ariz. (in California), several adults and nymphs (Glen M. Kohls); A. P. 17868, rock crevices in mine tunnel, May 24, 1940, Picacho Mountain near Picacho, Ariz., numerous adults and nymphs (Glen

M. Kohls); A. P. 17017, bat cave, July 2, 1940, near Freedom, Okla., two nymphs, one larva (D. E. Howell).

Ornithodoros yumatensis n. sp.

Body.—Sides nearly parallel, a little pointed in front, and broadly rounded behind. Pattern of the mammillae continuous over the margins without change of pattern from dorsal to ventral surfaces. Size of holotype female 4.75 mm.×3.25 mm. Size of allotype male 3.8 mm.×2.25 mm. Largest and smallest females 5.6 mm.×3.5 mm. to 4.1 mm.×2.25 mm. Largest and smallest males 4.8 mm.×2.40 mm. to 3.6 mm.×2.0 mm.

Mammillae.—Moderate in size and number and about equal in sizes in the median and peripheral areas on both dorsal and ventral surfaces; close, but not crowded. Individual mammillae on the dorsum are conical and have irregular surfaces with irregular radial ridges. Short hairs, few in number, present mainly on the posterior and lateral borders. Individual mammillae on the venter are bent backward, mildly suggesting reptilian scales.

Discs.—Present but not conspicuous; moderate in size, superficial or very mildly depressed. Present also on the venter in lineal arrangement in the preanal, transverse postanal, and the median postanal grooves.

Legs.—Long and slender, with their surfaces made irregular by very numerous fine granulations. Dorsal subapical protuberances and dorsal humps absent. Leg hairs are numerous and small, except on the distal portions of the tarsi, where they are longer. Length of female tarsus I, 1.02 mm.; metatarsus 0.72 mm.; tarsus IV, 1.17 mm.; metatarsus 0.96 mm. Length of male tarsus I, 0.66 mm.; metatarsus 0.51 mm.; tarsus IV, 0.87 mm., metatarsus 0.72 mm.

Coxae.—Coxae I and II a little separated, all others contiguous.

Hood.—Indefinite. The anterior point of the dorsal body wall may represent the anterior end of the hood, but if this is true the hood is short and small.

Camerostome.—Indefinite.

Movable cheeks.—Large, about twice as long as wide.

Capitulum.—The capitulum is protrusile and when extended reaches beyond the anterior end of the body. When so extended, the basis capituli is seen to be twice as long as wide and its length is about equal to the length of the soft membrane which unites the capitulum with the body. Living specimens may show the capitulum either extended or withdrawn. Specimens preserved in alcohol which are not enlarged by recent feeding have the capitulum withdrawn. Basis capituli with the surface granular (as on the legs) and faintly wrinkled. Numerous hairs present at the sides on the anterior portion (not

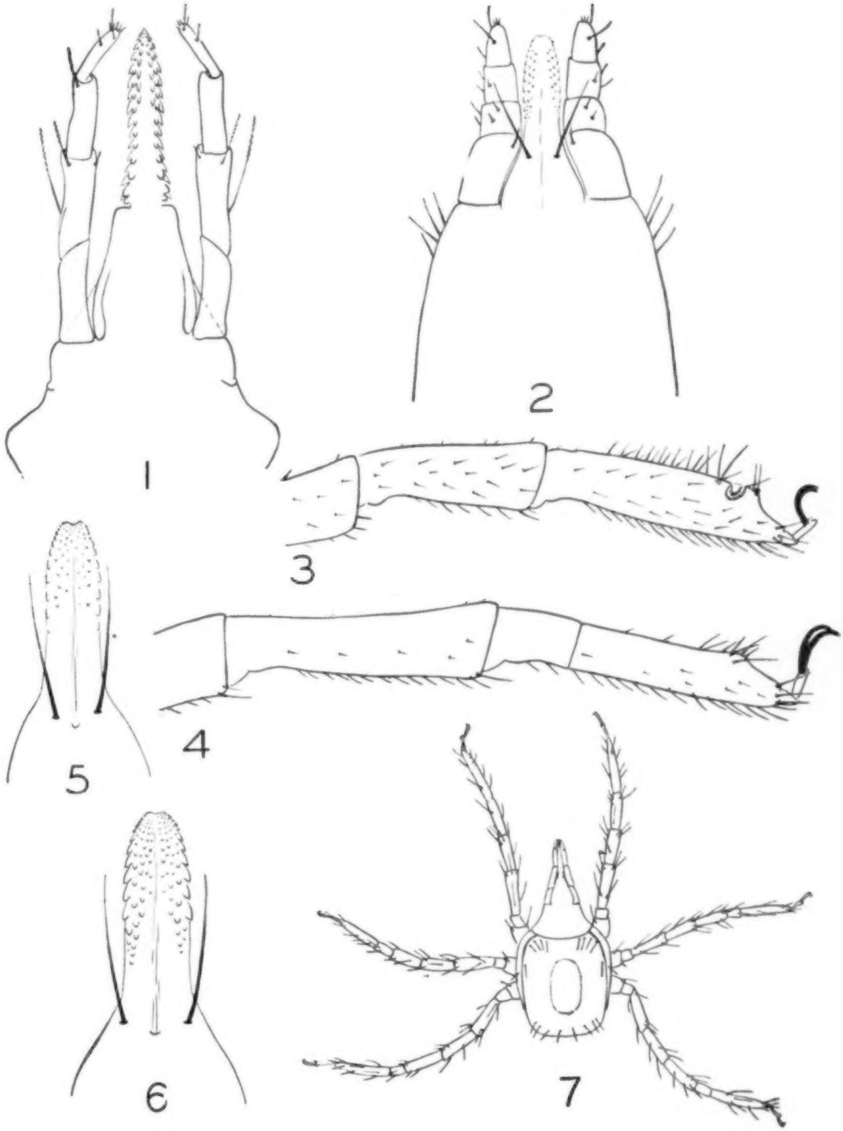


FIGURE 3.—*Ornithodoros yumatensis* n. sp. 1. Capitulum of larva, ventral view. 2. Capitulum of nymph, ventral view. 3. Tarsus and metatarsus of leg I of adult. 4. Tarsus and metatarsus of leg IV of adult. 5. Male hypostome. 6. Female hypostome. 7. Larva, dorsal view.

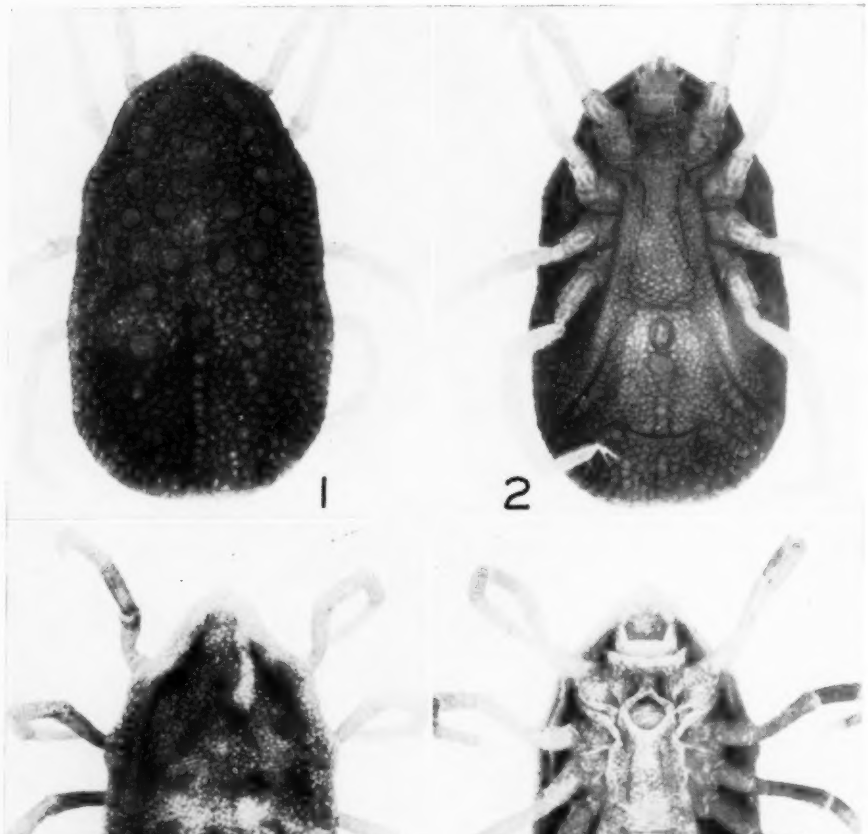


FIGURE 4.—1. *Ornithodoros stageri* n. sp., dorsal view. 2. *Ornithodoros stageri* n. sp., ventral view. 3. *Ornithodoros yumatensis* n. sp., dorsal view. 4. *Ornithodoros yumatensis* n. sp., ventral view. 5. *Ornithodoros eremicus* n. sp., dorsal view, nymph. 6. *Ornithodoros eremicus* n. sp., ventral view, nymph.

visible when the capitulum is withdrawn). Palpi tapering and with numerous hairs similar to those on the basis capituli. Tip of the hypostome reaching to the distal end of palpal article 3.

Hypostome.—Sides subparallel, apex notched. In both sexes the denticles are small or faint, largest in the lateral files and progressively smaller toward the median line. Length of hypostome, female 0.19 mm.; male 0.17 mm.

Folds.—Coxal and supracoxal folds present.

Grooves.—Preanal, transverse postanal, and median postanal grooves present. Dorso-ventral groove absent.

Sexual opening.—At the level of the interval between coxae I and II.

Eyes.—Absent.

Anus.—Large, in an elliptical pattern.

LARVA

Large, suboval. Length (including mouth parts) 1.20 mm., width 0.50 mm. Dorsal reticulated area is oval. Legs long, slender, and with claws. Capitulum terminal, large, its length equal to about half the entire length of the larva. Basis capituli large, visible from above, wider behind. Hypostome slender, long, pointed apically. Hypostome on a conical base which is about as long as the hypostome. Denticles apically, 3/3, then 2/2; those of the lateral (marginal) files large, those of the median files very small. Length of hypostome, 0.24 mm.

This species is rather closely related to *O. brodyi* Matheson 1935, but may be separated by the dentitions of the hypostomes and the characters of the individual mammillae. In the new species the latter are conical and have obvious radiating ridges on their sides, while in *brodyi* they are much flattened, smooth on top, and lack the radiating ridges.[2]

Holotype.—Female from A. P. 17881.

Allotype.—Male from A. P. 17881.

Paratypes.—25 females and 25 males from A. P. 17881.

Holotype female, allotype male, and paratypes at the Rocky Mountain Laboratory, except that single males and females have been deposited as follows: United States National Museum, Washington, D. C.; Zoological Division, Bureau of Animal Industry, Department of Agriculture, Washington, D. C.; Los Angeles County Museum, Los Angeles, Calif.; Division of Entomology and Parasitology, University of California, Berkeley, Calif.; Museum of Comparative Zoology, Harvard University, Cambridge, Mass.; Department of Entomology, Cornell University, Ithaca, N. Y.; Division of Entomology and Economic Zoology, University of Minnesota, Minneapolis, Minn.

[2] In our studies of the numerous species of *Ornithodoros* in North America in the preparation of a monograph now nearing completion, we have found that the individual mammillae show little variation and are very dependable as characters.

The three known collections, all by Glen M. Kohls, are from California and Arizona as follows: A. P. 17856, from rock crevices in mine tunnel, May 20, 1940, 15 miles northeast of Yuma, Ariz. (in California), one male, two nymphs; A. P. 17881, from rock crevices in Crystal Cave, June 2, 1940, 10 miles southeast of Winkelman, Ariz., numerous adults and nymphs, few larvae; A. P. 17882, from bats, *Myotis velifer velifer*, in Crystal Cave, June 2, 1940, 10 miles southeast of Winkelman, Ariz., six adults and nymphs.

CONTEMPLATED REVISION OF THE TREASURY DEPARTMENT DRINKING WATER STANDARDS

The requirements for drinking (and culinary) water provided by common carriers for the use of passengers carried in interstate traffic, commonly known as the "Treasury Department Drinking Water Standards," were last revised in 1925 and published in the Public Health Reports of April 10 of that year. Since that time many improvements in water supply practice have been adopted with resulting increased uniformity of quality and safety to the consumer. The revision of the standards to conform more closely to current requirements for water supplies of attainable safety and potability is accordingly in order.

To carry out such a revision the Surgeon General has appointed a special advisory committee composed of representatives of various Federal organizations and scientific associations and several members at large. A smaller subcommittee of Public Health Service officers has been designated to prepare tentative suggestions for changes in the present standards which will be submitted for the consideration of the advisory committee.

The membership of the advisory committee, together with the name of the organization which each represents, is as follows:

American Chemical Society:
A. M. Buswell,
Chief, Illinois State Water Survey Division,
Urbana, Ill.

American Public Health Association:
Abel Wolman,
Professor of Sanitary Engineering,
Johns Hopkins University,
Baltimore, Md.

American Society of Civil Engineers:
Arthur E. Gorman,
Bureau of Engineering,
Department of Public Works,
Chicago, Ill.

American Water Works Association:
Charles R. Cox,
Chief, Bureau of Water Supply,
State Department of Health,
Albany, N. Y.

Society of American Bacteriologists:
A. C. Hunter,
Principal Bacteriologist,
Food and Drug Administration,
Federal Security Agency,
Washington, D. C.

Conference of State Sanitary Engineers:
Arthur D. Weston,
Director and Chief Engineer,
Division of Sanitary Engineering,
State Department of Health,
Boston, Mass.

Food and Drug Administration:
J. W. Sale,
Senior Chemist, Food Division,
Federal Security Agency,
Washington, D. C.

U. S. Geological Survey:
W. D. Collins,
Chemist in Charge,
Washington, D. C.

Association of American Railroads:
R. C. Bardwell,
Superintendent, Water Supply,
Chesapeake & Ohio Railway,
Richmond, Va.

Member at Large:
R. F. Goudey,
Sanitary Engineer,
Bureau of Water Works and Supply,
Los Angeles, Calif.

Member at Large:
R. E. Buchanan,
Director, Agricultural Experiment Station,
Iowa State College,
Ames, Iowa.

Member at Large:
Herman G. Baity,
Professor of Sanitary Engineering,
University of North Carolina,
Chapel Hill, N. C.

United States Public Health Service:
Chairman:
Joseph W. Mountin,
Assistant Surgeon General,
Domestic Quarantine Division.
Secretary:
J. K. Hoskins,
Chief, Sanitation Section,
Domestic Quarantine Division.

The subcommittee, officers of the Public Health Service, consists of the following members:

H. W. Streeter, Senior Sanitary Engineer, Stream Pollution Investigations, Cincinnati, Ohio.

C. C. Ruchhoft, Principal Chemist, Stream Pollution Investigations, Cincinnati, Ohio.

C. T. Butterfield, Principal Bacteriologist, Stream Pollution Investigations, Cincinnati, Ohio.

Lawrence T. Fairhall, Principal Industrial Toxicologist, National Institute of Health, Bethesda, Md.

R. E. Tarbett, Senior Sanitary Engineer, Domestic Quarantine Division, Washington, D. C.

Secretary:
J. K. Hoskins, Senior Sanitary Engineer, Chief, Sanitation Section, Domestic Quarantine Division, Washington, D. C.

URBAN HOUSING AND CROWDING [1]

On the basis of Health Survey data covering 631,429 households in 83 cities and towns in various geographic areas in the United States and within the recognized limitations of the index, crowding in urban households as measured by number of persons per room has been found to vary widely in extent and in intensity with geographic region, color, size of household, and with tenancy, type, rental charge, or estimated value of dwelling units. The problem was found not to be limited to large cities; it was present in communities of all size groups and to varying degrees in cities of like size in the same geographic area. When measured in terms of persons the percentage of crowding was higher than when measured in terms of households. The pro-

portion of young children living under crowded conditions was much greater than the corresponding proportion in other age groups.

Regional differences in crowding may be summarized in the general statement that crowded households were relatively much more frequent in the South than in other areas; the lowest percentage of crowded households was found in the West; the North Central region was most typical of national conditions; and the Northeast showed less crowding than the North Central area. There were relatively fewer crowded white households in each household-size group in the Northeast than the West, although the rate for all white households was lowest in the West.

Patently, colored homes were more subject to crowded conditions than were white. In every comparison made, the crowding rate for the colored significantly exceeded that for the white. Not only was the percentage of crowded colored households higher in all specific population groups, but the intensity, as measured by the differentials in the proportions in the three degree-of-crowding categories, was greater among the colored. While proportionately 2½ times as many colored as white households were in dwellings with more than one person per room, the ratio of colored to white households with more than two persons per room was 4 to 1.

Crowding showed a direct association with household size and dwelling rental or value. The percentage of crowded units among smaller households was nominal except in the very low, as indicated by dwelling rental or value, economic status groups. In the minimum rental or value groups a notable proportion of the smaller households showed crowding. The larger households were found to include a high proportion living under crowded conditions even among the higher rental or value groups. This was particularly true of households in rented dwellings.

The proportion of crowded households in rented dwellings was (depending on the degree-of-crowding category) from 2½ to 5½ times as great as in owner-occupied dwelling units. A wide difference between the relative frequency of crowded households in rented dwellings of the multiple and of the single type was apparent, the single type of dwelling unit being less frequently crowded than the multiple.

Since large households were much more frequently crowded, the percentage of persons living under crowded conditions was greater than the percentage of households so domiciled; furthermore, for this reason and since large households were likely to include younger

---

[1] Britten, Rollo H., and Brown, J. E.: Urban housing and crowding: Relation to certain population characteristics as indicated by National Health Survey data. Public Health Bulletin No. 261. Available from the Superintendent of Documents, Government Printing Office, Washington, D. C., at 15 cents per copy.

children, the proportion of young children in crowded dwellings exceeded the proportion of persons in other age groups in crowded homes.

COURT DECISION ON PUBLIC HEALTH

Action by miner for injuries claimed to have resulted from inadequate ventilation of mine.—(Minnesota Supreme Court; *Applequist* v. *Oliver Iron Mining Co.*, 296 N.W. 13; decided January 17, 1941, rehearing denied January 30, 1941.) An action was brought by a miner to recover damages for personal injuries claimed to have been caused by the defendant mining company's failure to provide or maintain an adequate system of ventilation in its underground mines where he had been employed for several years. It was claimed that the miner had contracted "pneumoconiosis, sometimes called silicosis, and a serious lung ailment with other complications."

The statute relating to mines had its origin in a 1905 law, one section of which provided that, in case the inspector of mines found that a place was dangerous from any cause, it was his duty immediately to order the men engaged in work at the said place to quit work, to notify the person in charge to secure the place from the existing danger, and to specify the work to be done or change to be made to render the same secure, ordinary mine risks excepted. The so-called ventilating statute was first enacted in 1919 and one section of it provided that "all places of employment," as used in the law, should mean any place, either inside or outside, where any business or industry was carried on and in which persons were employed and should include, among other things, engineering works, but should not be construed to apply to domestic service or agricultural labor.

One question passed on by the Supreme Court of Minnesota was whether the ventilating statute was applicable in the instant case, the defendant claiming that the said statute did not apply to underground mining but that the mining statute alone applied. The appellate court, however, did not agree with the defendant's contention, saying that it thought that the ventilating statute was an addition to and an enlargement of the duties cast upon industry in general, for the obvious purpose, so far as practicable, of protecting those employed by it; that it applied to underground mining; and that where, as in the instant case, an underground miner became afflicted with a disabling ailment, not covered by the compensation act, through negligence of the employer amounting to the omission of a statutory duty, viz, the failure of the employer properly to ventilate an underground mine where the employee worked, such employee had an action at law for damages.

DEATHS DURING WEEK ENDED MARCH 8, 1941

From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Mar. 8, 1941	Corresponding week, 1940
Data from 88 large cities of the United States:		
Total deaths	9, 101	9, 365
Average for 3 prior years	9, 383	
Total deaths, first 10 weeks of year	96, 749	96, 048
Deaths under 1 year of age	548	460
Average for 3 prior years	526	
Deaths under 1 year of age, first 10 weeks of year	5, 488	5, 306
Data from industrial insurance companies:		
Policies in force	64, 655, 691	66, 069, 866
Number of death claims	13, 532	15, 103
Death claims per 1,000 policies in force, annual rate	10. 9	12. 0
Death claims per 1,000 policies, first 10 weeks of year, annual rate	10. 9	10. 7

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED MARCH 15, 1941

Summary

The only item of significance in the current weekly reports of the 9 communicable diseases included in the following table is the incidence and increase in the number of cases of measles. A total of 43,060 cases was reported for the current week, as compared with 34,420 for the preceding week and with 31,490 for the next earlier week. The current figure may be compared with 43,622 cases reported for the corresponding week in 1938, the highest number reported for the week since 1935. In 1938, the latest "measles year," the peak of 44,191 cases was reached during the week ended March 26.

The Middle Atlantic, East North Central, and South Atlantic States continue to report the highest incidence of measles, and the New England, Middle Atlantic, West South Central, and Pacific areas reported more cases for the current week than for the corresponding week of 1938.

The current figures for only measles and whooping cough were above the 5-year (1936–40) median, while those for diphtheria, scarlet fever, smallpox, and typhoid fever were below those reported for the corresponding week in each of the preceding 5 years. Only 11 cases of poliomyelitis were reported, of which 3 occurred in Florida. Of 65 cases of smallpox, 55 cases were reported in 8 States of the North Central areas, while no cases were reported in the New England, Middle Atlantic, or South Atlantic States. One case of Rocky Mountain spotted fever was reported in Montana and 1 case of undulant fever in Utah. One case of endemic typhus fever was reported in Oregon, and 23 cases were reported from the Southern States.

The death rate for the current week for 92 major cities in the United States was 12.7 per 1,000 population, the same as for the preceding week. The 3-year (1938–40) average for 88 cities is 12.8.

Telegraphic morbidity reports from State health officers for the week ended March 15, 1941, and comparison with corresponding week of 1940 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended		Median 1936-40	Week ended		Median 1936-40	Week ended		Median 1936-40	Week ended		Median 1936-40
	Mar. 15, 1941	Mar. 16, 1940		Mar. 15, 1941	Mar. 16, 1940		Mar. 15, 1941	Mar. 16, 1940		Mar. 15, 1941	Mar. 16, 1940	
NEW ENG.												
Maine	0	2	2	1	12	12	94	356	202	0	1	1
New Hampshire	0	0	0	3	------	------	23	30	30	0	0	0
Vermont	0	0	0	------	------	------	18	4	46	0	0	0
Massachusetts	2	2	3	------	------	------	811	311	864	2	0	2
Rhode Island	0	0	0	------	------	------	5	138	82	1	1	1
Connecticut	1	1	2	4	7	18	61	156	156	0	1	1
MID. ATL.												
New York	12	15	34	[1] 54	33	[1] 38	7,605	383	1,408	3	2	3
New Jersey	10	4	11	36	23	23	2,549	280	280	0	0	1
Pennsylvania	17	11	40	------	------	------	4,958	40	322	7	7	7
E. NO. CEN.												
Ohio	6	12	24	98	217	48	5,704	17	252	2	2	3
Indiana	21	6	12	39	61	61	627	8	14	1	0	2
Illinois	24	21	35	44	35	35	4,152	113	70	4	3	3
Michigan [2]	3	9	9	33	23	5	4,416	178	178	1	5	0
Wisconsin	0	2	2	200	224	67	838	267	267	0	1	1
W. NO. CEN.												
Minnesota	0	14	4	29	1	1	11	179	179	0	0	0
Iowa	6	2	3	136	28	8	175	196	133	0	0	1
Missouri	6	20	16	11	16	253	151	9	13	1	0	1
North Dakota	2	6	2	21	44	44	13	6	6	0	0	0
South Dakota	0	1	0	6	2	2	27	1	2	0	0	0
Nebraska	2	0	2	15	------	4	6	107	46	0	1	1
Kansas	2	11	9	13	31	40	661	533	20	1	0	0
SO. ATL.												
Delaware	0	0	0	------	------	------	351	4	32	0	0	0
Maryland [3]	3	5	5	41	57	57	170	3	199	1	1	2
Dist. of Col	5	6	7	5	------	3	126	5	39	1	0	0
Virginia	14	10	20	1,077	552	552	1,971	44	220	2	3	3
West Virginia [2]	10	4	9	125	610	218	338	17	17	1	1	2
North Carolina [3]	7	16	13	83	8	172	921	141	167	0	0	1
South Carolina [3]	6	2	5	754	774	872	278	7	37	1	0	0
Georgia [3]	9	11	9	257	144	286	421	254	205	2	1	2
Florida [3]	6	1	8	159	9	9	973	92	92	0	0	3
E. SO CEN.												
Kentucky	4	3	12	135	69	93	1,317	25	110	1	0	6
Tennessee	4	6	9	161	238	416	339	95	165	2	0	5
Alabama [3]	6	14	8	316	335	1,862	481	124	124	2	3	7
Mississippi [2,3]	8	3	4	------	------	------	------	------	------	2	0	1
W. SO. CEN.												
Arkansas	9	7	7	291	334	334	238	36	36	0	0	0
Louisiana	5	1	12	76	62	62	61	26	26	2	1	1
Oklahoma	12	3	6	207	491	343	14	7	12	2	1	1
Texas [3]	29	36	36	1,167	1,761	1,677	767	811	475	0	1	4
MOUNTAIN												
Montana [4]	7	0	1	11	11	25	5	31	18	1	0	0
Idaho	1	0	0	------	2	4	44	39	13	0	0	0
Wyoming	1	0	0	------	5	------	19	19	19	0	0	0
Colorado	10	6	8	44	29	------	214	30	30	0	0	0
New Mexico	5	1	4	5	7	21	187	37	37	0	0	1
Arizona	0	2	2	105	224	224	136	95	57	0	0	0
Utah [2]	0	2	1	22	8	------	32	315	105	0	0	0
Nevada	0	------	------	------	------	------	0	------	------	1	------	------
PACIFIC												
Washington	2	1	1	16	11	5	79	653	257	1	0	1
Oregon	1	10	1	21	31	72	442	421	45	0	0	0
California [3]	13	26	30	404	211	211	231	533	609	1	3	3
Total	291	315	458	6,225	6,740	8,852	43,060	7,176	11,626	46	39	66
11 weeks	3,233	4,379	5,956	556,322	140,504	85,103	222,361	59,774	80,967	499	428	1,009

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended March 15, 1941, and comparison with corresponding week of 1940 and 5-year median—Continued

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40
	Mar. 15, 1941	Mar. 16, 1940		Mar. 15, 1941	Mar. 16, 1940		Mar. 15, 1941	Mar. 16, 1940		Mar. 15, 1941	Mar. 16, 1940	
NEW ENG.												
Maine	0	0	0	7	11	17	0	0	0	0	0	0
New Hampshire	0	0	0	3	0	7	0	0	0	0	0	0
Vermont	0	0	0	8	8	8	0	0	0	0	0	0
Massachusetts	0	1	0	168	102	287	0	0	0	1	2	2
Rhode Island	0	0	0	5	18	18	0	0	0	0	0	0
Connecticut	0	0	0	52	89	130	0	0	0	1	0	0
MID. ATL.												
New York	0	2	2	524	1,049	1,049	0	0	0	5	3	4
New Jersey	0	0	0	330	358	239	0	0	0	1	3	1
Pennsylvania	0	2	1	338	257	514	0	0	0	6	6	6
E. NO. CEN.												
Ohio	0	1	1	256	343	343	0	0	1	2	4	2
Indiana	0	0	0	191	275	264	5	1	5	1	1	1
Illinois	0	0	1	516	870	870	14	2	13	0	2	4
Michigan [2]	0	2	0	252	383	442	3	0	2	0	3	3
Wisconsin	1	1	0	166	153	186	7	3	5	1	0	1
W. NO. CEN.												
Minnesota	0	0	0	59	88	135	5	1	9	0	1	1
Iowa	0	0	0	65	62	233	1	4	23	0	1	1
Missouri	0	0	0	86	102	216	19	2	8	4	5	2
North Dakota	0	0	0	21	14	29	0	6	6	0	0	0
South Dakota	0	0	0	11	5	16	0	1	4	0	0	0
Nebraska	0	0	0	24	20	30	0	1	11	1	0	0
Kansas	0	1	0	52	67	189	1	0	14	0	0	1
SO. ATL.												
Delaware	0	0	0	16	17	6	0	0	0	0	0	
Maryland [2]	0	0	0	36	42	47	0	0	0	1	2	1
Dist. of Col.	0	0	0	32	18	18	0	0	0	0	0	0
Virginia	0	0	0	48	36	36	0	0	0	1	2	3
West Virginia [2]	1	0	0	47	50	58	0	0	0	2	0	2
North Carolina [3]	0	1	0	34	26	41	0	0	1	0	0	1
South Carolina [3]	0	0	0	5	3	3	0	0	0	10	2	1
Georgia [3]	0	0	0	14	17	17	0	5	0	3	3	3
Florida [3]	3	0	0	8	5	10	0	0	0	4	1	2
E. SO. CEN.												
Kentucky	0	1	0	151	94	68	0	0	0	2	1	2
Tennessee	1	0	0	153	81	55	1	1	0	3	2	2
Alabama [3]	1	0	0	26	23	15	1	0	0	5	2	2
Mississippi [2] [3]	0	0	0	6	5	5	2	1	0	1	2	2
W. SO. CEN.												
Arkansas	1	1	1	7	2	10	0	3	2	3	2	2
Louisiana	1	1	0	14	14	13	0	1	3	5	9	9
Oklahoma	0	0	0	13	15	25	0	27	15	1	5	2
Texas [3]	0	3	1	58	39	94	2	2	5	2	3	14
MOUNTAIN												
Montana [4]	1	0	0	38	26	28	0	0	9	0	0	0
Idaho	0	0	0	11	10	22	0	0	3	[5] 9	1	1
Wyoming	0	0	0	11	6	10	0	0	0	1	0	0
Colorado	0	0	0	51	29	57	1	10	6	0	0	0
New Mexico	0	0	0	5	13	30	0	2	0	0	8	2
Arizona	0	0	0	3	10	10	0	0	0	2	0	1
Utah [2]	0	0	0	16	27	35	0	1	1	0	1	0
Nevada	0	------	------	1	------	------	1	------	------	0	------	------
PACIFIC												
Washington	0	0	0	12	53	47	0	0	10	0	3	2
Oregon	0	1	0	11	24	39	2	0	18	1	3	1
California [3]	1	2	2	170	193	239	0	2	18	5	5	5
Total	11	20	20	4,131	5,152	6,205	65	76	327	84	88	101
11 weeks	317	307	228	38,482	51,089	67,405	522	810	3,297	[5] 833	827	1,213

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended March 15, 1941, and comparison with corresponding week of 1940 and 5-year median—Continued

Division and State	Whooping cough	
	Week ended	
	Mar. 15, 1941	Mar. 16, 1940
NEW ENG.		
Maine	13	32
New Hampshire	11	5
Vermont	7	35
Massachusetts	227	171
Rhode Island	18	6
Connecticut	66	29
MID. ATL.		
New York	318	319
New Jersey	128	86
Pennsylvania	402	225
E. NO. CEN.		
Ohio	421	235
Indiana	25	47
Illinois	83	92
Michigan [2]	351	188
Wisconsin	97	102
W. NO. CEN.		
Minnesota	95	23
Iowa	51	5
Missouri	65	31
North Dakota	8	3
South Dakota	14	0
Nebraska	7	3
Kansas	102	57
SO. ATL.		
Delaware	5	5
Maryland [3]	72	210
Dist. of Col	9	15
Virginia	98	52
West Virginia [2]	55	62
North Carolina [3]	340	108
South Carolina [3]	123	14
Georgia [3]	83	9
Florida [3]	18	5
E. SO. CEN.		
Kentucky	102	51
Tennessee	59	40
Alabama [3]	36	31
Mississippi [2, 3]	-------	-------
W. SO. CEN.		
Arkansas	11	2
Louisiana	2	30
Oklahoma	21	3
Texas [3]	256	208
MOUNTAIN		
Montana [4]	28	5
Idaho	19	28
Wyoming	0	3
Colorado	57	6
New Mexico	16	53
Arizona	20	14
Utah [2]	92	123
Nevada	2	-------
PACIFIC		
Washington	84	61
Oregon	7	30
California [3]	463	241
Total	4,587	3,103
11 weeks	46,813	31,804

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Typhus fever, week ended March 15, 1941, 24 cases as follows: North Carolina, 2; South Carolina, 2; Georgia, 9; Florida, 4; Alabama, 2; Mississippi, 2; Texas, 2; California, 1.
[4] Rocky Mountain spotted fever, week ended March 15, 1941, Montana, 1 case.
[5] Delayed report has been received of the earlier occurrence of 13 cases of typhoid fever in Idaho.

WEEKLY REPORTS FROM CITIES

City reports for week ended March 1, 1941

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5 year average	139	948	137	5, 381	917	2, 085	29	394	20	1, 120	--------
Current week[1]	90	598	84	13, 319	555	1, 369	3	377	18	1, 146	--------
Maine:											
Portland	0	--------	0	5	7	3	0	1	0	12	21
New Hampshire:											
Concord	0	--------	0	0	0	1	0	0	0	0	9
Manchester	0	--------	0	0	1	2	0	0	0	0	11
Nashua	0	--------	0	0	0	1	0	0	0	1	9
Vermont:											
Barre	--------	--------	--------	--------	--------	--------	--------	--------	--------	--------	--------
Burlington	0	--------	0	0	0	0	0	0	0	0	10
Rutland	0	--------	0	0	2	0	0	0	0	0	7
Massachusetts:											
Boston	1	--------	2	239	8	44	0	10	0	93	226
Fall River	0	--------	0	0	1	19	0	0	0	6	28
Springfield	0	--------	0	1	1	0	0	0	0	1	40
Worcester	0	--------	0	104	6	4	0	0	0	13	53
Rhode Island:											
Pawtucket	0	--------	0	0	0	0	0	0	0	0	15
Providence	0	2	1	4	9	4	0	1	0	12	67
Connecticut:											
Bridgeport	0	--------	0	1	6	4	0	2	0	4	37
Hartford	1	--------	0	1	3	1	0	4	1	5	49
New Haven	0	2	0	0	0	13	0	0	0	7	54
New York:											
Buffalo	0	--------	2	54	12	23	0	5	0	16	139
New York	17	68	2	4, 252	96	256	0	89	4	98	1, 632
Rochester	0	--------	0	15	4	1	0	1	0	2	60
Syracuse	0	--------	0	0	2	0	0	1	0	9	53
New Jersey:											
Camden	2	1	0	35	2	9	0	4	0	5	20
Newark	0	11	0	251	8	47	0	3	0	21	115
Trenton	0	3	0	17	2	64	0	2	0	0	46
Pennsylvania:											
Philadelphia	2	4	1	1, 462	35	109	0	34	1	51	529
Pittsburgh	0	5	1	92	11	10	0	10	0	56	184
Reading	0	--------	0	299	4	0	0	2	0	5	36
Scranton	0	--------	--------	1	--------	1	0	--------	0	0	--------
Ohio:											
Cincinnati	5	7	3	77	7	14	0	5	0	2	144
Cleveland	0	48	3	1, 981	15	49	0	8	0	65	223
Columbus	1	1	1	62	1	19	0	2	0	25	107
Toledo	0	5	3	22	8	0	0	2	0	4	84
Indiana:											
Anderson	0	--------	0	0	1	0	0	0	0	0	18
Fort Wayne	0	--------	0	38	4	1	0	1	0	0	31
Indianapolis	2	--------	0	89	16	24	0	3	0	3	114
Muncie	0	--------	0	3	3	13	0	0	0	0	17
South Bend	0	--------	0	10	2	1	0	0	0	0	15
Terre Haute	0	--------	1	0	2	1	0	0	0	0	18
Illinois:											
Alton	0	--------	0	0	0	3	0	0	0	0	8
Chicago	16	18	5	1, 913	33	203	0	29	2	37	710
Elgin	1	--------	0	32	3	0	0	0	0	0	8
Moline	0	--------	0	10	0	0	0	0	0	1	10
Springfield	1	--------	0	0	4	6	0	0	0	2	28
Michigan:											
Detroit	6	16	3	980	12	129	0	13	1	123	303
Flint	1	--------	0	38	6	6	0	0	0	10	29
Grand Rapids	0	--------	1	97	1	6	0	0	0	9	37
Wisconsin:											
Kenosha	0	--------	0	61	0	0	0	0	0	4	6
Madison	0	--------	0	14	0	3	0	0	0	3	20
Milwaukee	0	1	1	81	10	24	0	0	0	41	120
Racine	0	--------	0	5	0	7	0	0	0	2	14
Superior	0	--------	1	0	0	5	0	0	0	6	9

[1] Figures for Barre and Boise estimated; reports not received.

City reports for week ended March 1, 1941—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Minnesota:											
Duluth	0	--------	1	0	1	1	3	0	0	3	23
Minneapolis	0	11	2	0	3	14	0	1	0	27	112
St. Paul	0	1	1	1	1	4	0	3	1	11	58
Iowa:											
Cedar Rapids	0	--------	--------	0	--------	1	0	--------	0	0	--------
Davenport	0	--------	--------	0	--------	0	1	--------	0	0	--------
Des Moines	0	--------	--------	0	--------	8	0	--------	0	1	35
Sioux City	0	--------	--------	0	--------	3	0	--------	0	9	--------
Waterloo	0	--------	--------	1	--------	1	0	--------	0	1	--------
Missouri:											
Kansas City	0	--------	1	5	7	20	0	5	0	11	92
St. Joseph	1	--------	0	3	3	0	0	0	0	1	22
St. Louis	3	6	3	46	27	68	0	14	0	32	260
North Dakota:											
Fargo	0	--------	0	0	1	1	0	0	0	7	3
Grand Forks	0	--------	--------	0	--------	0	0	--------	0	0	--------
Minot	0	--------	--------	1	--------	0	0	--------	0	5	3
South Dakota:											
Aberdeen	0	--------	--------	0	--------	1	0	--------	0	0	--------
Sioux Falls	1	--------	--------	0	--------	0	0	--------	0	0	10
Nebraska:											
Lincoln	0	--------	--------	2	--------	7	0	--------	0	0	--------
Omaha	0	--------	1	1	5	3	0	1	0	1	61
Kansas:											
Lawrence	0	2	0	62	1	0	0	0	0	0	3
Topeka	0	--------	0	65	1	4	0	1	0	3	7
Wichita	0	2	0	0	5	1	0	0	0	20	31
Delaware:											
Wilmington	0	--------	0	161	0	2	0	0	0	1	33
Maryland:											
Baltimore	0	13	3	24	25	30	0	15	0	60	249
Cumberland	0	--------	0	0	1	0	0	0	0	0	12
Frederick	0	--------	1	0	0	0	0	0	0	0	4
Dist. of Col.:											
Washington	2	15	3	67	10	11	0	13	0	11	178
Virginia:											
Lynchburg	0	--------	0	2	0	1	0	0	1	0	13
Norfolk	0	77	2	58	2	2	0	1	0	0	25
Richmond	0	--------	4	13	6	2	0	0	0	0	49
Roanoke	0	--------	--------	262	--------	2	0	--------	0	2	15
West Virginia:											
Charleston	0	6	0	75	0	2	0	0	0	1	19
Huntington	0	--------	--------	0	--------	0	0	--------	0	0	--------
Wheeling	0	--------	0	0	4	0	0	1	0	3	20
North Carolina:											
Gastonia	0	--------	--------	5	--------	0	0	--------	0	7	--------
Raleigh	0	--------	1	43	6	0	0	0	0	23	13
Wilmington	0	--------	0	0	3	0	0	0	0	0	15
Winston-Salem	0	10	2	2	2	2	0	3	0	19	25
South Carolina:											
Charleston	0	42	0	45	3	0	0	2	2	0	19
Florence	0	--------	1	0	5	0	0	0	0	0	18
Greenville	0	--------	0	49	9	1	0	0	0	11	32
Georgia:											
Atlanta	1	10	6	14	3	5	0	1	0	2	96
Brunswick	0	1	1	2	1	1	0	0	0	1	5
Savannah	0	172	4	3	1	2	0	2	0	0	44
Florida:											
Miami	0	10	1	11	2	0	0	2	2	7	57
Tampa	0	1	0	1	2	1	0	0	0	0	39
Kentucky:											
Ashland	0	1	0	0	2	0	0	1	0	0	9
Covington	0	1	0	34	0	0	0	0	0	1	15
Lexington	0	--------	0	4	5	0	0	0	0	1	19
Louisville	0	16	0	74	3	52	0	5	0	15	60
Tennessee:											
Knoxville	0	1	1	20	2	5	0	1	0	5	29
Memphis	0	1	3	38	6	4	0	6	0	17	94
Nashville	0	--------	1	21	6	4	0	1	0	3	51
Alabama:											
Birmingham	1	65	1	63	7	1	0	4	0	4	69
Mobile	1	1	2	5	1	1	0	0	0	0	23
Montgomery	0	3	--------	21	--------	2	0	--------	0	1	--------
Arkansas:											
Fort Smith	0	2	--------	4	--------	0	0	--------	0	0	--------
Little Rock	0	--------	3	1	5	4	0	5	0	1	47

City reports for week ended March 1, 1941—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Louisiana:											
Lake Charles	0		0	0	0	0	0	0	0	0	4
New Orleans	2	5	4	1	13	4	0	13	0	10	156
Shreveport	1		0	0	4	1	0	4	1	0	50
Oklahoma:											
Oklahoma City	0	13	0	0	8	1	0	0	0	0	40
Tulsa	0		0	0	5	3	0	0	0	11	34
Texas:											
Dallas	6	1	1	7	4	8	0	1	1	5	84
Fort Worth	1		7	78	6	3	0	0	0	1	33
Galveston	0		0	0	2	0	0	2	0	0	21
Houston	2	1	1	0	10	2	0	7	0	0	97
San Antonio	2	6	1	0	5	0	0	3	0	5	63
Montana:											
Billings	0		0	0	0	1	0	0	0	0	4
Great Falls	0		0	1	2	2	0	1	0	0	10
Helena	0		0	1	0	0	0	0	0	0	4
Missoula	0		0	0	3	0	0	0	0	0	8
Idaho:											
Boise											
Colorado:											
Colorado Springs	0		0	3	0	3	0	1	0	1	10
Denver	8	11	0	44	3	5	0	4	2	28	95
Pueblo	0		0	1	1	1	0	0	0	1	13
New Mexico:											
Albuquerque	0	1	0	8	0	0	0	0	0	0	5
Utah:											
Salt Lake City	0		0	3	1	1	0	1	1	5	29
Washington:											
Seattle	4		0	3	3	8	0	4	0	11	101
Spokane	0	1	1	5	2	1	0	0	0	0	40
Tacoma	0		0	0	2	0	0	1	0	5	42
Oregon:											
Portland	0	1	0	21	1	4	0	1	0	0	79
Salem	0			1		0	0		0	1	
California:											
Los Angeles	0	37	3	20	10	24	0	13	0	34	356
Sacramento	1		0	1	2	4	0	2	0	11	37
San Francisco	0	2	1	5	11	9	0	12	0	22	233

State and city	Meningitis, meningococcus		Poliomyelitis cases	State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
Connecticut:				Maryland:			
Bridgeport	1	1	0	Baltimore	1	1	
New York:				District of Columbia:			
New York	1	2	1	Washington	1	1	1
Pennsylvania:				Georgia:			
Philadelphia	1	0	0	Atlanta	0	0	1
Scranton	1	0	0	Kentucky:			
Ohio:				Covington	0	0	1
Cleveland	1	0	0	Oklahoma:			
Michigan:				Oklahoma City	1	0	0
Detroit	1	0	0	Texas:			
Minnesota:				Galveston	1	0	0
St. Paul	1	0	0				

Encephalitis, epidemic or lethargic.—Cases: New York, 4; Pittsburgh, 1; Minneapolis, 1; Denver, 1.
Pellagra.—Cases: Kansas City, Mo., 1; Savannah, 1.
Typhus fever.—Cases: Savannah, 2; Dallas, 1. Deaths: Los Angeles, 1.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended February 8, 1941.—During the week ended February 8, 1941, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis		5	2	4	14	1	1	2	5	34
Chickenpox		7		102	307	30	16	18	88	568
Diphtheria		16		12	1	5	5			39
Dysentery				8						8
Influenza		54			184	9			76	323
Measles		261	164	364	756	190	603	438	934	3,710
Mumps				119	126	20	5	10	34	314
Pneumonia		15			13	4	1		30	63
Poliomyelitis				1						1
Scarlet fever		26	4	119	147	4	7	20	4	331
Tuberculosis	2	6	14	58	37	2	1			120
Typhoid and paratyphoid fever				16	4					20
Whooping cough				153	140	6	4	7	21	331

FINLAND

Communicable diseases—4 weeks ended December 31, 1940.—During the 4 weeks ended December 31, 1940, cases of certain communicable diseases were reported in Finland as follows:

Disease	Cases	Disease	Cases
Diphtheria	211	Poliomyelitis	23
Influenza	14,508	Scarlet fever	341
Paratyphoid fever	201	Typhoid fever	64

SWEDEN

Notifiable diseases—December 1940.—During the month of December 1940, cases of certain notifiable diseases were reported in Sweden as follows:

Disease	Cases	Disease	Cases
Diphtheria	47	Scarlet fever	921
Dysentery	1	Syphilis	23
Epidemic encephalitis	3	Typhoid fever	6
Gonorrhea	756	Undulant fever	12
Paratyphoid fever	18	Weil's disease	1
Poliomyelitis	19		

YUGOSLAVIA

Notifiable diseases—4 weeks ended December 29, 1940.—During the 4 weeks ended December 29, 1940, certain notifiable diseases were reported in Yugoslavia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	8	1	Paratyphoid fever	19	1
Cerebrospinal meningitis	90	30	Poliomyelitis	1	
Diphtheria and croup	581	47	Scarlet fever	257	7
Dysentery	107	13	Sepsis	10	3
Erysipelas	133	7	Tetanus	21	11
Favus	21		Typhoid fever	374	42
Lethargic encephalitis	2	2	Typhus fever	5	

×